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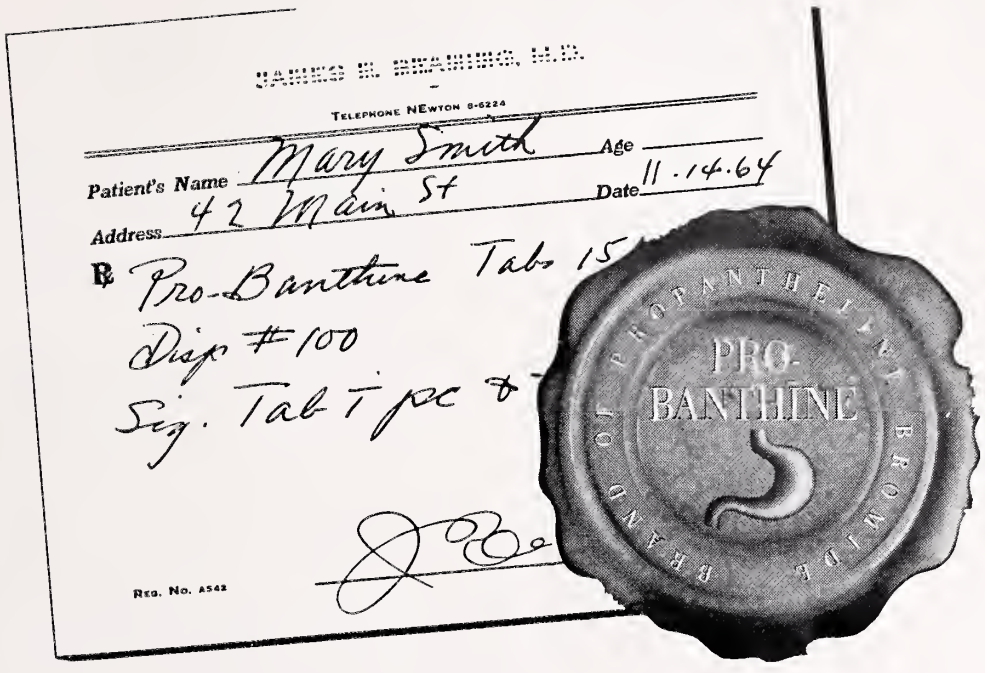
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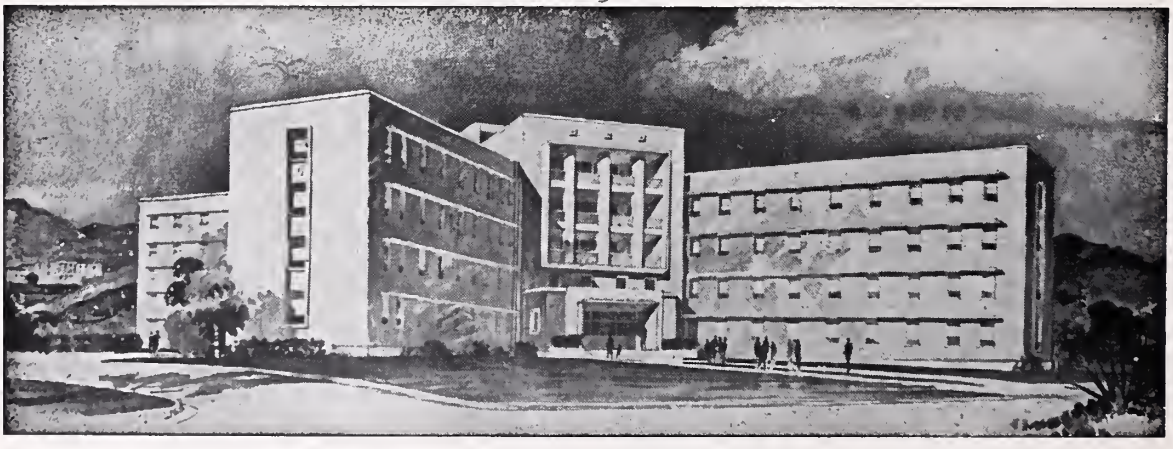
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Health Care — Present and Future

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Public Relations Chairmen for county societies are also invited, as well as all members of the state society and their wives. The meeting will be held at Bishop's Lodge.

Legislation, national and state, will be discussed and a blueprint outline for each society to use in opposing and supporting these legislative measures will be presented. Reports on three special meetings in Chieago sinee the AMA meeting in Miami will be given. Dr. David G. Clark of Albuquerque is State Public Relations Chairman.

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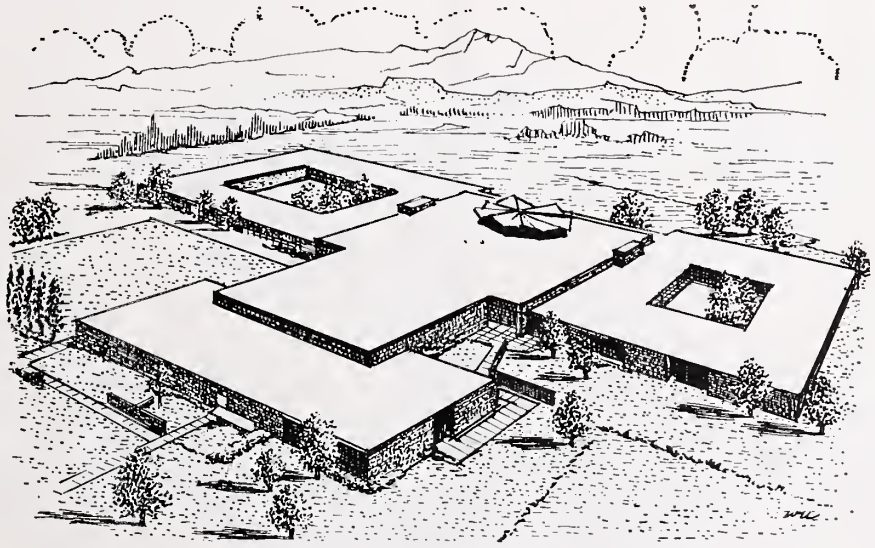
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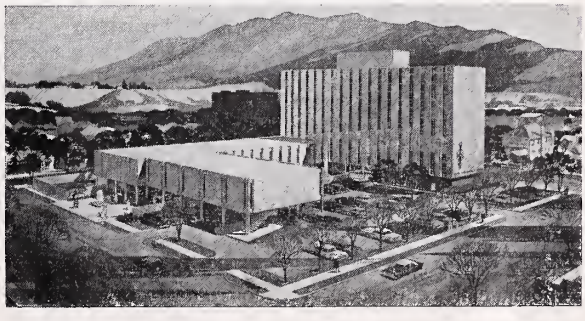
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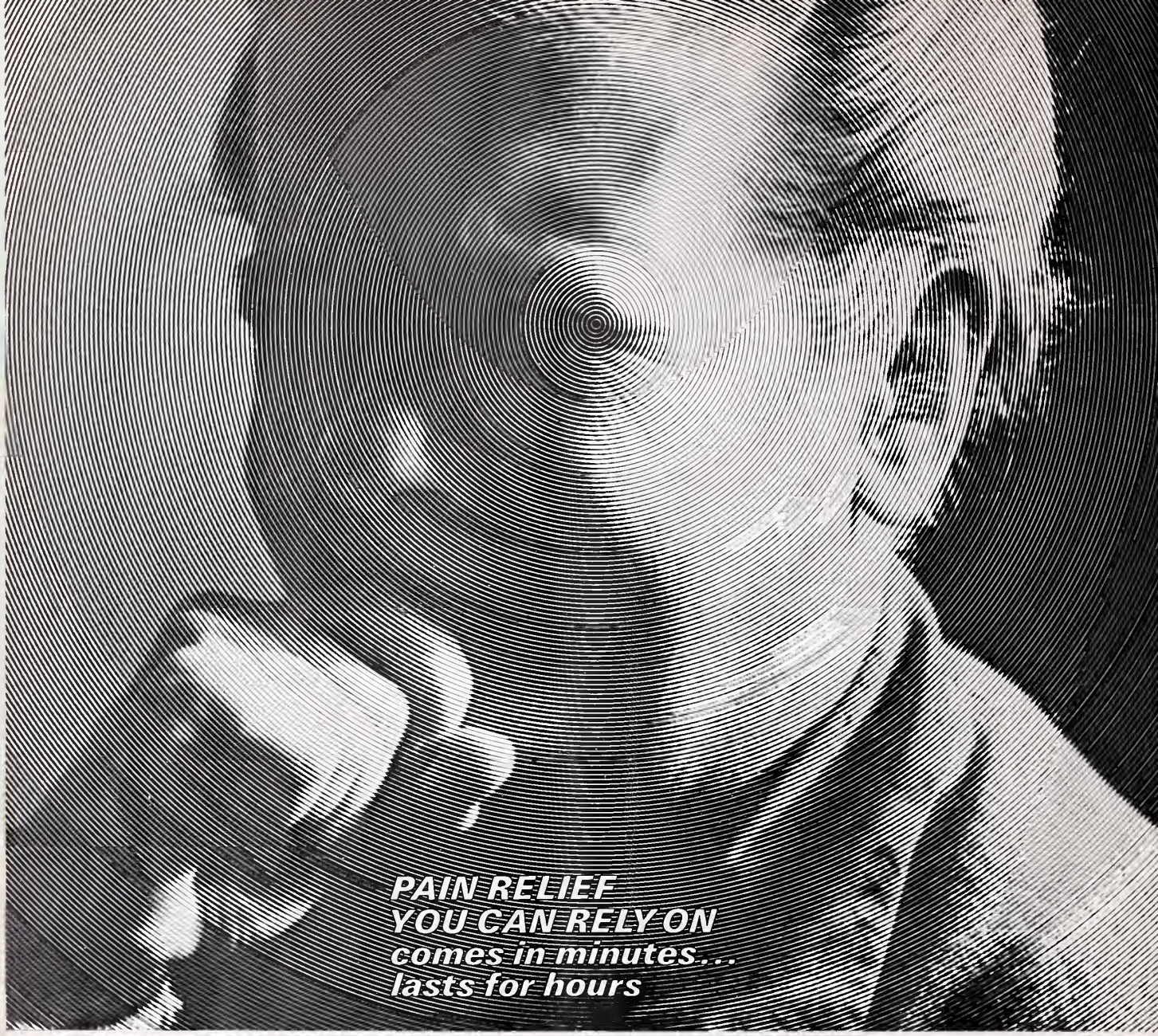
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*Roseman, E.: *Neurology* **11**:912, 1961. 33664

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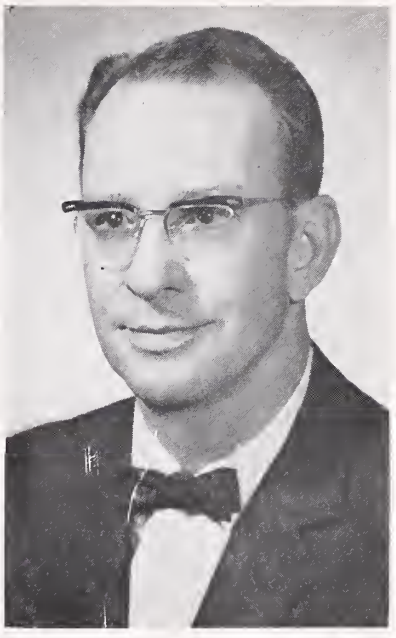
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Dr. Boverie Elected President of El Paso County Medical Society



Dr. Robert F. Boverie, long active in the Southwestern Medical Association, was elected President of the El Paso County Medical Society for the 1964-65 year at the annual meeting of the Society on December 8, 1964. Dr. H. D. Garrett was the retiring President.

Other new officers are Dr. J. T. Bennett, President-Elect; Dr. W. W. Schuessler, a Past President of the Southwestern Medical Association, Vice-President; Dr. L. N. Nickey, Secretary; Dr. B. B. Kern, Secretary-Elect; and Dr. W. L. Pierce, Treasurer.

Born in Wellington, Texas, Dr. Boverie received a B.A. from Texas Technological College and his M.D. from the University of Texas Medical Branch at Galveston. He interned and then took a four-year residency in Radiology at Western Reserve University Hospital in Cleveland.

He is certified by the American Board of Radiology, has a Fellowship degree from the American College of Radiology, and is Councilor for Western Texas for the Radiological Society of North America.

He is a member of the Texas Radiological Society, Rocky Mountain Radiological Society, Pan-American Radiological Society, Society of Nuclear Medicine, Western Association of Railway Surgeons, is an honorary member of the Southwestern Obstetrical and Gynecological Society, and is a Civilian Consultant in Radiology at William Beaumont Army Hospital.

He is Past President of the Hotel Dieu Hospital Staff, El Paso Chapter of the American Cancer Society, El Paso Catholic Physicians Guild, the El Paso Unit of Serra International, and the El Paso Diocesan Council of Catholic Men.

He has served on the National Executive Board of the National Council of Catholic Men, the Board of Directors of the Medical Center Corporation in El Paso, the Diocesan Board of Catholic Charities, and is at present on the board of the El Paso Chapter of the American Cancer Society. He is a member of St. Joseph's Church. He was made a Knight of St. Gregory by Pope Pius XII.

He has been practicing medicine in El Paso since 1946 with the group now known as Drs. Hart, Boverie, Black, Clayton, Green and White.

Dr. and Mrs. Boverie reside at 4430 Oxford Avenue with four daughters and two sons. A daughter Judy is a student in the University of Madrid in Spain.

Asymptomatic Bacteriuria with Pregnancy

(A Review of the Literature)

Robert J. Oliver, M.D.,* *Tucson*

Introduction

Perinatal mortality programs are established all over the country in a concentrated effort to reduce the death rate by prophylaxis of maternal diseases. Certain causes of fetal and neonatal death are "controlled", but over 50 per cent of fetal loss is still inexplicable. Prematurity, occurring in 10-12 per cent of all births accounts for a large number of deaths (500-1000 gms.—20%).[5] Of prematurity, 14-15 per cent are due to maternal diseases: chiefly, toxemia, placenta previa; abruptio placenta. Ten to 12 per cent are multiple gestations. The remainder are idiopathic premature labor.[5] These areas are under close scrutiny by many clinicians. Renal disease is one area of intense investigation.

An imposing amount of scientific literature in the past three years has been devoted to urinary tract disease and its relationship with pregnancy. Previously, investigators have speculated that bacilliuria should be looked for and treated only when symptomatic. Now, however, with the improved techniques in the laboratory and in pharmacology, concerned obstetricians are questioning the advisability of ignoring asymptomatic bacteriuria of pregnancy.

The purpose of this review is: 1) To find the incidence and significance of bacteriuria in pregnancy; 2) its relationship to the "routines" of obstetrical management; 3) to study its relationship to perinatal morbidity, prematurity, and puerperial complications: 4) its influence on future disease in the female.

*Formerly a resident in the Department of Obstetrics and Gynecology, Mercy Hospital, San Diego, California, Thomas L. Slate, M.D., Director of Obstetrics and Gynecology Residency.

Occurrence

The overall incidence of bacteriuria in pregnancy falls between six per cent and 12.5 per cent in all studies reviewed. Turck[21] reported his study of 1,727 patients. By dividing the total into groups, he found two per cent occurence in whites and 8.6 per cent in Negroes, with 6.5 per cent in the lower socio-economic groups. In the latter, 11.4 per cent were multiparas. Multiparity was related to a much higher incidence of bacteriuria in all other studies, and more will be said in this regard later.

Turner[23] reported 105 antenatal patients with asymptomatic, but significant, bacteriuria. She found that of these, 60 per cent developed symptomps before delivery. Others[8, 11, 14] have likewise noted asymptomatic cases becoming symptomatic in the antepartum period; not, however, in that high a ratio unless other factors were present, such as trauma, catheterization, or retention of urine.

In a recent report from the University of California,[14] seven per cent of 1,400 patients had significant bacteriuria prenatally, and when retested six months postpartum, bacteria had disappeared from the urine in one-third of the patients receiving no treatment. Thirteen per cent of their cases had become symptomatic in that six month period. Significant bacteriuria continued into the puerperium in two-thirds of Boswell's series. He and many others[4, 1, 12, 17, 14, 25] have proposed that asymptomatic contamination of the urine can and often does continue from one pregnancy to the next, becoming more potentially dangerous with each succeeding pregnancy. Renal

pathophysiologists are becoming increasingly concerned since adequate follow-up may detect this contamination and by adequate antibacterial agents reduce the incidence of pyelonephritis as age advances.

Clinical Picture

Symptomatic bacteriuria is a concise clinical entity and will not be discussed here.

The use of quantitative colony counts has been exercised with great success in the establishment of significant bacteriuria in the female. Benign vaginal discharges in purulent leukorrhea, not to mention the usual bacterial contaminations around the urethral orifice present difficulties in obtaining "clean" urine without catheterization. Many clinicians continue the argument that it cannot be done. In reading the many carefully prepared studies using the "clean-catch" midstream method for specimen collection, there remains little doubt of its efficacy for urine examination and culture.[7] The urethral tract has been shown to contain large numbers of bacteria and even careful catheterization will carry organisms beyond the urethra and into the bladder.[15] This area, the bladder, is felt to be the area of colonization.[14]

In our clinic, the patient is instructed to spread the labia, and using aqueous zephrin solution, cleanse the area surrounding the urethral meatus thoroughly; then in a squatting position obtain the mid-portion of the voided specimen. This has been quite successful in reducing the amount of contamination which we had previously noted. There are many variations of the technique, all adequately described; each can be easily adapted to office use. The aloquate is then placed in appropriate culture medium and at 24 and 48 hour intervals quantitative counts are done. Kass[10] felt significant bacteriuria was present when 100,000 colonies per ml. were found — less than this was felt to represent contamination. Occasionally, symptomatic bacteriuria of less than 100,000 colonies per ml. is found,[14] but this is rare.

A new color sensitive test is now available and gives results in two to four hours. This has been found to be most efficacious although no identification can be made.

Causes of Bacteriuria in Pregnancy

Recognized as one of the most important pathophysiological causes for the increased incidence of renal disease in the pregnant female is purely anatomicophysiological. Cruveilhier described this phenomena in 1841. Dilatation of the ureters and renal pelvis occurs as a result of, 1) progesterone — leading to the loss of smooth muscle tone and diminished peristalsis, 2) pressures imposed at the pelvic brim (especially on the right), due to the weight of the gravid uterus,[5] and 3) vesico-urethral reflux.[1, 25] These changes are found to be present by the third month, increasing with each trimester. This leads to stasis of urine and increased chance for bacterial colonization. Author after author has shown the non-catheterized patient has bacteriuria from 0-5 per cent; the catheterized patient, regardless of the reason for catheterization has two to three times more urinary contamination. The more she is "relieved" of her urine, the greater the chance of contamination. This fact alone is responsible for the majority of the renal infections found in our patients, whether antepartum, postpartum, pre-operatively, or postoperatively. For routine use, the catheter has been discarded completely, or at least replaced by metal or glass. Except for obstetrical or surgical indications and urinary retention, catheterization is found to be unnecessary and frequently harmful. Significant bacteriuria is two times more common in diabetics than in general population.[6] This is probably due in large part to the high levels of glucose spilled in their urine. Anemia is found to be a cause and result of symptomatic bacteriuria. Correction of the anemia also increases the resistance of the body to invasion of the urinary tract.

Relationship of Prenatal Morbidity, Prematurity, and the Puerperium

"Pyelitis" is felt to be an ascending infection, implicating again the bladder as a petredish. Pyelitis of pregnancy with its discomfort, high fever, and more serious implications is the most common complication of pregnancy, occurring in five to 10 per cent of all pregnancies. From Kass' study, 60 per cent of the asymptomatic bacteriuric patients became infected during the course of the pregnancy — most of these in the last trimester.[7,8] In Monzon's report,[14] this did not hold true, since they found most symptomatic cases in the latter

half of the first and all of the second trimesters — few in the last. This was particularly true in the untreated cases of asymptomatic bacteriuria. Therefore, by adequate detection of the asymptomatic bacteriuria and its treatment **before** invasion occurs, a very common and troublesome syndrome can be avoided.

Postpartum, the asymptomatic bacteriuria disappears in approximately one-third of the cases within six months, without treatment; however, 10-15 per cent become symptomatic in the puerperium if untreated.[21, 22, 8, 14] Pyelonephritis resulting from this insult to the urinary tract is the most serious acute and chronic complication found with bacteriuria. Kass' catheterized patients developed "apparent" pyelonephritis in 40 per cent of the untreated cases, but no pyelonephritis in the treated group. He made the statement, "elimination of bacteriuria eliminated pyelonephritis". His findings are substantiated by a great many investigators — some find 50 per cent and as high as 65 per cent — depending upon one's interpretation of the diagnosis of pyelonephritis. The great danger, other than the sepsis and toxic reactions of that disease, is its chronicity and, for the obstetrician, hypertensive disease.

Pregnancy imposes an increased renal excretory load, which is an added stress in the presence of glomerulonephritis, pyelonephritis, and/or hypertensive disease. Obviously then, we must be on the lookout for this disease, which can be actively dormant in the "preeclamptic patient" with bacteriuria. Reversing this slightly, in a large series of patients studied by Switzer,[20] women with pyelonephritis and bacteriuria predominated over men; however, no correlation of hypertension and bacteriuria could be found, but was suggested with hypertension and pyelonephritis. Asymptomatic bacteriuria is not benign.

Cause or effect? Monzon[14] and Pinkerton[17] in doing random radiological studies on their patients **with** bacteriuria found 41-43 per cent congenital (hypoplatic, usually) kidney disease. This figure is higher than the proposed incidence should be and is striking. Further study might well be done along these lines. Of their group, 55 per cent of those with kidney deformity had symptomatic bacteriuria whereas only 25 per cent had had no previous symptoms. Radiological changes consistent with pyelonephritis were found in 35 per cent of the patients with renal deformity.

We, as obstetricians, have an opportunity to reduce a disease which kills just as surely as does cancer.

Treatment

Briefly, since treatment is well established, the therapy of symptomatic bacteriuria is bacteria-specific antibiotics: the sulfonamides, -cyclines, and nitrofurantoin derivatives. Prevention of retention, and maintenance of proper renal flow are of paramount importance. In the resistant case, adequate study including cystoscopy, radiological examination, and bacteriological study are imperative.

The asymptomatic case should be treated first prophylactically — avoidance of unnecessary catheterization, obstetrical trauma, and prevention of stasis. Appropriate antibiotics should be given in time long enough to keep the urine abacteric; therefore, the careful examiner will recheck the urine of his patient frequently and culture at the first sign of microscopic evidence of bacteria.

Postpartum prevention of or clearing of bacteria from the urine should be the task of each attending physician.

Prematurity

What influence has asymptomatic bacteriuria on the outcome of a single pregnancy? We know from previous experience the results of severe acute pyelonephritis, or for that matter, any renal disease in pregnancy — mostly bad; hence, I shall not waste paper or ink in this regard. I pause only to mention the syndrome of preeclampsia and prematurity, because acute and chronic pyelonephritis mimic these toxic diseases.[7, 1, 24]

In multiple studies of uterine contractility, excessive activity is recorded in any patient with pyelitis, cystitis and vaginitis. The endotoxins of coliform organisms are particularly irritating to the profused myometrium. Spontaneous premature labor appears to be characteristic in chronic renal disease. Bruns[3] felt that spontaneous premature rupture of the aforewaters was related to this excessive uterine activity, and at the Premature Center in Colorado, the urinary tract is vigorously treated.

Kass feels prematurity is two to three times more frequent in patients with bacteriuria than in those without contamination. Prematurity occured in 24 per cent of his group with bacteriuria, as compared with 10 per cent in controls. Also,

there was 17 per cent perinatal mortality as compared to **no** deaths in the two groups. Other investigators do not duplicate this feeling but cannot or do not dogmatically say it is not so. Monzon found no association in this regard. Bruns also felt no association existed in the asymptomatic group. This feature is under extensive study now; more results are soon to be published.

A casual relationship can be postulated only when a complication of pregnancy precedes premature labor or perinatal mortality more frequently than would be expected. Definite causes are found only 38 per cent of the time. Chronic hypertension, abruptio placenta, placenta previa, renal disease, multiple pregnancy, and toxemia make up most of this 38 per cent. No definite conclusions in regard to prematurity can be drawn from this review of bacteriuria, but will have to await more complete study.

Conclusion

The dangers of chronic infectious renal disease are well established in the studies reviewed. **No** report contradicts this statement. Bacteriuria can be a sign or a cause of existing disease in the excretory system. Our job is to use this finding as a tool in detection and prevention. The value of the "pap" smear is not denied. More deaths result from cardiovascular-renal disease each year than for any single cause — including cancer.

Summary

An attempt is made to review the recent literature in regard to bacteriuria associated with pregnancy. Several facts are notable:

1) Clean-catch specimen of urine with quantitative colony count of 100,000/ml., gram stain, or Urescreen indicates significant bacteriuria.

2) Routine catheterization, regardless of the technique or technician, results in contamination of the urinary tract and is to be avoided.

3. Adequate follow-up with repeated cultures and prompt therapy will reduce symptomatic changes and pyelonephritis in those with asymptomatic bacteriuria.

4) Bacteriuria as another cause of prematurity can be implicated, but only insofar as it becomes symptomatic.

The body's image is reflected in its urine.

910 North Wilmot Rd.

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Break-Through in Snakebite

W. E. Lockhart, M.D., *Alpine, Texas*

Heretofore snakebite treatment has not been effective. False optimism and favorable evaluation of treatment has been because of the fact that significant envenomization occurs in only half of the cases, and whatever treatment was used — whiskey, cautery, incision-suction or antivenin — was credited with success of the half without significant envenomization[1].

Ninety-six per cent of rattlesnake bites occur on the arm and leg[1]: therefore, the objective of treatment is to preserve function of the hand and foot. Approximately one per cent of rattlesnake bites are fatal, and death usually occurs several days after the bite[1].

Tissue destruction — destruction of muscle, nerve, blood vessels and tendons — is the result of proteolytic digestion by enzymes of the venom, which is a modified saliva. Therefore, any effective treatment must halt the proteolytic digestive action of the venom in the tissues. Massive doses of species specific antivenin given intravenously experimentally has proved to be NOT effective in halting the proteolytic action of the enzymes[2]. Excision, wide and deep, of the area of the bite is effective if carried out within one hour of the bite[3], but this treatment can be carried out only in sites where tissue is expendable and replaceable, as on the trunk, and cannot be used on valuable tissues, such as the arm and leg or hand and foot, and this treatment can be carried out only when the patient reaches the surgeon within an hour after the bite.

First-Aid treatment should consist of an immediate tourniquet applied proximal to the bite and left in place for one hour and then removed and not replaced. Much harm has been done by leaving a tourniquet in place too long. The victim should avoid all possible exertion, and transportation to a hospital should be provided. Incision and suction should NOT be administered by a layman because danger of infection and hemorrhage. Antivenin should NOT be administered by a layman because of the danger of anaphylaxis from horse serum hypersensitivity. The area of the bite and as much of the extremity as possible should be packed with fresh water ice held in place by a plastic sheet (such as a pillow protector) at the earliest possible time. Demerol and other opiates should be avoided because they add to the lethal effect of the venom[2], and antihistamines should be avoided for the same reason[1]. In half an hour numbness from the ice will relieve the pain.

Like any surgical procedure, cryotherapy or ice treatment of snakebite should be carried out with careful attention to detail, and certain contraindications should be observed. The contraindications are any condition causing impaired vascularity of the extremity such as: Raynaud's Disease, long delayed treatment with swelling and destruction of blood vessels, mutilating incisions with secondary infection or a tight tourniquet left in place too long. A tourniquet is never used simultaneously with cryotherapy. The ice must be fresh water ice that will not melt below ZERO degrees Centigrade. Addition of salt would result in freezing and frostbite and gangrene and must be avoided. As much as possible of the extremity is packed with crushed ice held in place with a plastic sheet, and there must be no obstruction to vascularity of the extremity. The remainder of the patient's body is kept "uncomfortably warm" by use of an electric blanket, and the patient should take abundantly of food, fluids and electrolytes[2]. This avoids chilling which would delay immunity response and venom destruction by warm blood components in the refrigerated extremity. The ice should be packed directly against the patient's skin.

The nurse should have specific instructions (1) To uncover the extremity very briefly every hour day and night and check the following: (a) Pink color of skin, (b) Good pulse, (c) Capillary circulation by pressing and releasing finger or toe nail, and (d) Temperature next to the skin measured with a chemical (Centigrade) ther-

mometer. (2) To immediately repack the extremity with an abundance of cracked ice and replace the plastic cover. Restlessness in a child may be controlled by a 25 mgm. rectal suppository of Thorazine.

Tetanus toxoid should be given to prevent tetanus. Specific antivenin may be given according to directions in the package but not before (1) Skin scratch test, (2) Conjunctival test and (3) Intradermal test (in that order) prove the absence of horse serum hypersensitivity. Antibiotics are given to prevent or control infection. Cortisone, since it does not inhibit proteolytic enzyme activity, is of no value.

Cryotherapy or ice treatment of snakebite is effective in all species of bites[4] and, indeed, all envenomizations. The doctor will be at a loss to know WHEN to discontinue cryotherapy. The usual signs used to judge the degree of envenomization, such as swelling, pain, redness, discoloration and blebs, are inhibited by cryotherapy. A severe rattlesnake envenomization will require SIX days and nights of CONTINUOUS cryotherapy. The ice treatment should NOT be interrupted too soon because this permits remaining enzymes of the venom to become active.

Report of two cases

Case 1. D. Y., Latin-American, female, age 4 years, was bitten by a "small snake that rattled" (presumably the "Prairie Rattler", Crotalus viridis viridis, known for extreme toxicity despite its small size) in the Big Bend National Park. Within a few minutes the foreleg was packed in fresh water ice, and she was brought to Brewster County Memorial Hospital, where the ice pack was maintained continuously for four days and nights. Tetanus toxoid, antivenin and antibiotics were given. On the fourth day the absence of fever, swelling, pain, redness or blebs misled me to believe that a mistake had been made and that possibly there had been on envenomization. But within an hour after the ice was discontinued fever shot up to 102 F., she was crying with pain. Next day there was swelling, redness and huge toxic skin blebs characteristics of rattlesnake bite. During the ice treatment the child was cheerful, ate well and played with her dolls, but now she was restless, crying, anorexic, toxic and febrile. Thus, **cryotherapy had completely inhibited the proteolytic enzymes of the venom for four days,** and subsequent events proved that severe envenomization had occurred. There is minor residual shortening of the flexor longus digitorum muscle.

Case 2. B. L. H., White, male, age 4 years was bitten by a "small snake that rattled" (Presumably Crotalus viridis viridis) in his back yard as he attempted to rescue a kitten from the snake. The two fang holes were evident on the knuckles of the right index and third fingers. He was packed immediately in fresh water ice and brought to Brewster County Memorial Hospital where ice treatment was continued for SIX days and nights without any interruption. Because of the extreme value of these tissue sites, no incisions or excisions were done. Tetanus toxoid, antivenin but no antibiotics were given. He had no fever. His body was kept warm with an electric blanket. The skin of the arm remained pink, the pulse good and capillary circulation in the finger nail bed was good throughout the treatment. Thorazine 25 mgm rectal suppository was given when needed for restlessness in the early hours of treatment. At the end of treatment full function of the hand resumed within an hour after discontinuation. There was residual swelling, redness and tenderness in the dorsum of the hand and forearm for several days. No disability resulted. Thus, **if there was significant envenomization in this case, the cryotherapy completely inhibited the proteolytic enzymes and prevented any tissue destruction. NO HARM WAS DONE TO THE EXTREMITY BY SIX DAYS CONTINUOUSLY OF FRESH-WATER ICE!**

In every case of snakebite two factors are uncontrollable: HOW MUCH and HOW TOXIC was the venom injected. In Case 1, above, the presence of severe, toxic envenomization was proved to be present by later events after premature discontinuation of the ice, and the inactivity of the venom during the four days of ice therapy proves that the enzymes were inhibited in the tissues at the lowered temperature. Case 2, above, proves that six days continuously in freshwater ice does no harm to a normal extremity.

Conclusion: Now at last we have a safe method of treatment for snakebite which will completely inhibit tissue destruction and prevent the horrible disability that heretofore we accepted as inevitable in some cases. Two cases are presented to substantiate the statement by Dr. Irving S. Cooper in Medical World News, September 25, 1964: "The

big advantage of cryosurgery is its reversability. In the temperature range of 15 degrees C. to 0 degrees C., tissue temporarily shows all the physiological inhibitory effects of freezing without producing permanent destruction". Subsequent careful clinical research should prove that cryotherapy or ice treatment for snake bite meets the first criterion of the American Academy of Science: that cryotherapy do no harm to normal tissues. Subsequent experience should also prove whether cryotherapy is more efficient than any previous treatment in preventing or reducing residual disability from snakebite.

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Foot Care and the Schools

Carlsbad Health Department

Do schools have a responsibility in the foot care of children, especially those in Junior and Senior High School? School personnel, particularly physical education teachers and coaches, feel that they do: classroom teachers believe that young people with itching, painful feet are not able to work at their maximum capacity. Many parents express the opinion that hygienic conditions in locker and shower rooms contribute to the prevalence of foot conditions referred to as athlete's foot. In Carlsbad, New Mexico, this mutual concern led to a study carried out by the public school personnel, the City Health Department*, the Medical Advisory Committee to the Health Department**, a Special Consultant in Podiatry***, and 1,733 Junior and Senior High School students. This study, which was made over a period of five months, resulted in the conclusion that the use of a medicated foot powder after bathing is the single, most important factor in the prevention of new cases of athlete's foot (tinea pedia) and allied conditions. Certain other measures were found to be essential to good foot hygiene which greatly influences the prevention and improvement of these skin conditions.

Each year thousands of students, as well as adults, experience uncomfortable skin conditions which are generally referred to as athlete's foot, and these are often extremely resistant to treatment. For years, such lesions were thought to be spread by pathogenic fungi picked up from the floors of shower rooms, bath rooms, and locker rooms, but study and experimentation indicate that fungous disease of the feet is probably not spread by such exposure. It is true, however, that feet clinically free from fungous disease often harbor pathogenic fungi, but attempts to demonstrate these fungi in shower rooms, etc. have been unsuccessful. Also, attempts to produce athlete's foot or fungous disease in healthy subjects by exposing the feet to heavy concentrations of pathogenic fungi in foot baths have failed. These studies**** support the position, stressed by many dermatologists and podiatrists, that clinical fungous disease is the result of decreased resistance rather than exposure.

At the outset of the Carlsbad study, a review of the literature revealed several gaps in knowledge that obviously needed to be filled before recommendations could be made to parents, physical education teachers, and coaches for the control of fungi by proper foot care. Since it was believed that many skin diseases of the feet result from decreased resistance on the part of the host and that good foot hygiene will aid in preventing decreased resistance, the study was undertaken to obtain information regarding decreased foot resistance and to define good foot hygiene. Therefore, the following questions were outlined:

1. Will the practice of washing feet with hexachlorophene soap during each daily shower be of significant benefit in eradicating athlete's foot among physical education students and athletes?

*Claude F. Tabor, B.S., M.P.H., Director

**Earl B. Flanagan, M.D., Internal Medicine; Ross A. Manganaro, M.D., Radiology. George B. Markle, IV, M.D., Surgery; Rupert H. Pate, M.D., Surgery

***Leon Cohen, D.S.C.

****Baer and Rodolph, American Journal of Public Health, June 1955

2. Will the after-shower use of a medicated powder on the feet decrease the incidence of skin disease of the feet?

3. What is the effect of the use of a chlorine disinfectant in foot baths?

4. What is the effect of wearing thongs in shower and locker rooms?

Questionnaires were distributed to physical education teachers and coaches who reviewed them with the students under their supervision. This resulted in 1,733 completed questionnaires which revealed the rather surprising information that approximately 25 per cent (433) had definite skin problems with their feet. This seemed a rather high proportion and one which certainly could not be ignored. Further tabulation showed that the girls who were participating in physical education classes and sports were not having the same percentage of problems as boys who were engaged in the same types of activities. Of the 894 boys interviewed, 37 per cent were having mild to severe skin problems with their feet while only 12 per cent of the 839 girls were experiencing difficulties of a similar nature. Faced with these facts, the need for a program of good foot hygiene in the Carlsbad School System appeared imperative.

Consideration was given to the difference in the number of problems among the girls and among the boys. The findings of the Medical Advisory Committee and the Consultant in Podiatry showed that girls had fewer problems. It was presumed that this was because (1) many girls wear no stockings; (2) most girls wear lighter shoes and lower shoes than boys; (3) when sitting in classes or elsewhere girls frequently take off their shoes or slip their heels out of their shoes, all of which tend to minimize excessive perspiration of the feet. Perspiring feet are moist feet, and moist feet are an excellent environment for the growth and multiplication of fungi. Because of the smaller number of problems, it was decided that, since skin diseases of the feet were much more prevalent among the male students than among the girls, only the boys would be studied. Four study groups and four control groups were formed from 521 students, all being of similar age, economic status, and environment.

While the Health Department surveyed the environmental conditions associated with each group, the members of the Medical Advisory Committee and the Consultant in Podiatry examined each student to determine that actual skin disease was present and the type. As the program developed, voluminous, accurate information was compiled.

In the control groups, the students took showers daily, but did nothing in addition to prevent foot problems.

Study Group I—**Medicated Soap**[1]: Medicated soap (hexachlorophene) was used to wash the feet thoroughly during each shower.

Study Group II—**Medicated Powder**[2]: Medicated powder was used after each daily shower.

Study Group III—**Foot Baths with Disinfectant:** The students in this group walked, after each shower, through a foot bath containing a disinfectant (200 parts per million free available chlorine).

Study Group IV—**Thongs**: The students in this group wore thongs at all times while in dressing and shower rooms.

Study Group	% New Problems During 5 Month's Study
Medicated Soap	6.98
Medicated Powder	1.67
Foot Bath with Disinfectant	12.12
Thongs	18.75

As shown in the preceding table, the single most important factor found to prevent new cases of athlete's foot and allied conditions was **the use of medicated powder[2] after each daily shower.**

In the control group, a composite shows that the incidence of new skin problems at the end of the study period was 20.8 per cent as shown below.

Control Group	Number	% New Problems During 5 Month's Study	
Medicated Soap	54	9.68	
Medicated Powder	91	11.67	
Foot Bath with Disinfectant	49	25.00	20.8
Thongs	54	34.78	

While the primary objective of the Carlsbad study was to determine the best preventive measures that could be established by school officials, many other factors were revealed to physicians and health authorities as they worked together to resolve this question. One very important factor was that the regular use of medicated soap[1] during each daily shower was extremely useful in improving some of the known cases of skin diseases of the feet.

Other factors revealed were:

Students who wore cotton socks had fewer problems than those wearing wool and stretch socks.

Oxfords are preferable to boots, sneakers, or tennis shoes for all-day wear.

The wearing of thongs and the use of chlorine disinfectant in foot baths seemed to be of some value in improving and preventing skin diseases of the feet but did not compare favorably with the use of medicated powder and medicated soap.

Recommendations for Increased Resistance to Diseases of the Feet

1. Keep feet clean by washing them with medicated soap.[1]

2. Use (antiseptic) medicated foot powder[2] after thorough drying of feet following bathing.

3. Wear light weight, light colored socks to minimize excessive perspiration. Change socks whenever they become damp.

4. Wear the shoes that cause the least perspiration.

5. Physical education teachers and coaches should frequently review with students the importance of carrying out these recommendations and should consider this a part of the education program.

6. Parents should be informed of these findings and recommendations, and their cooperation should be enlisted.

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1. The medicated soap (Dial) was supplied by Armour and Co.
2. The medicated foot powder (Desenex) was supplied by WTS Pharmaceuticals.

Please address all correspondence regarding this manuscript to: E. B. Flanagan, M.D., 906 West Pierce St., Carlsbad, New Mexico 88220

Announce Program For District One Postgraduate Meeting

Dr. Robert S. Nelson, Association Professor of Medicine (Gastroenterology) at the University of Texas Postgraduate School of Medicine, and a member of the Staff of the University of Texas M. D. Anderson Hospital and Tumor Institute, will be one of three speakers at a postgraduate session, February 7, 1965, at a two-day annual meeting of District One of the Texas Medical Association in Pecos, Texas, starting February 6.

Dr. Nelson will talk on "Liver Disease, and The Evaluation of Liver Function Tests" and "Infectious Hepatitis." Also on the postgraduate program are Dr. John A. Shively and Dr. Fred B. Kessler, both of Houston. Dr. Shively is Pathologist and Chief of the Section of Clinical Pathology at The University of Texas M. D. Anderson Hospital and Tumor Institute. He will speak on "Evaluation of Laboratory Data and Clinical Diagnosis" and "Bone Marrow Failure and Myelofibrosis." Dr. Kessler is Clinical Instructor in Surgery at the Baylor University College of Medicine. His subject will be "Evaluation and Immediate Care of the Injured Hand" and "Reconstruction of the Injured Hand." The postgraduate program is being provided by the University of Texas Graduate School of Biomedical Sciences and is under the direction of Dr. J. Leighton Green of El Paso.

Both sessions of the District One meeting will be at the Pecos Country Club, where Dr. Max E. Johnson, San Antonio, President of the TMA, will speak at a luncheon, February 6, opening the annual assembly. The scientific program that afternoon will run from 2 to 4:40 p.m. and will be followed by a business meeting at 4:50 p.m. and a cocktail supper at 6 p.m. Sunday's session will start at 9 a.m. and close at 5:15 p.m.

Speakers and their subjects at the first scientific session Saturday will be Dr. John M. Verosky, El Paso, "Vector Cardiography in Children"; Dr. George W. Iwen, El Paso, "Esophagal Lesions Seen in a Community Hospital"; Dr. Jean Turner Bowman, El Paso, "Treatment of Metastatic Carcinoma of the Breast"; and Dr. Dale F. Rector, El Paso, "pH and pCO_2." Dr. William R. Gaddis of El Paso is program chairman. Registration fee will be $20.

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Teen-age food consumption patterns are studded with missed meals, erratic eating habits, food faddism, fear of fatness, and culinary conformity to the group. Teen-age food choices frequently reflect a misguided independence expressed as a defiance of good food habits inculcated in childhood. Teen-age girls in their zest for stylish slenderness and clear complexions have the least nutritious diets of any member of the family. As prospective mothers and homemakers they are most in need of nutritional guidance yet most resistant to advice and counsel unless properly motivated. Teen-age boys in their desire for athletic prowess and muscular magnificence are more interested in food quantity than in food quality. Ironically, teen-age nutritional omissions are rarely the result of privation. They stem instead from an ignorance of, or indifference to, the virtues of good nutrition.

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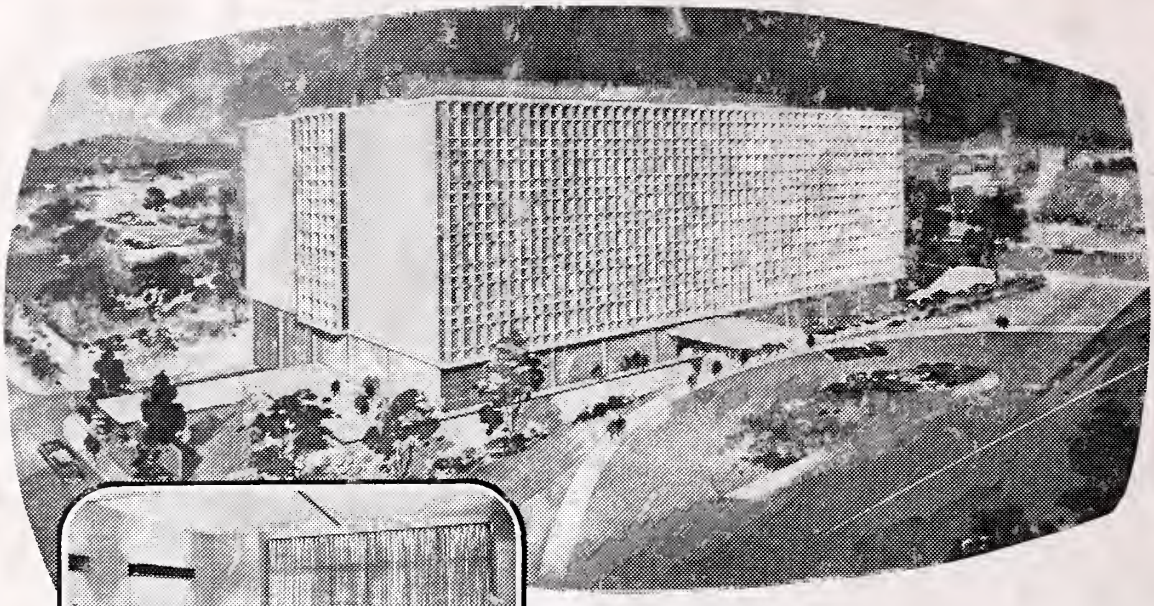
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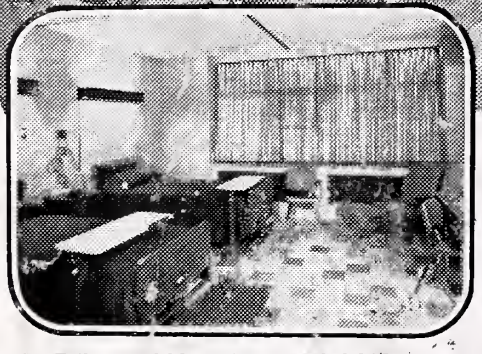
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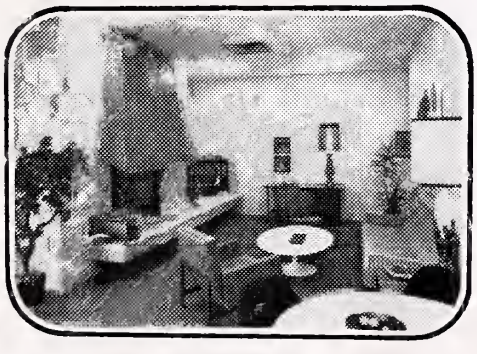
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1. Griffith, R. S., and Black, H. R.: Current Ther. Res., *6*:253, 1964.

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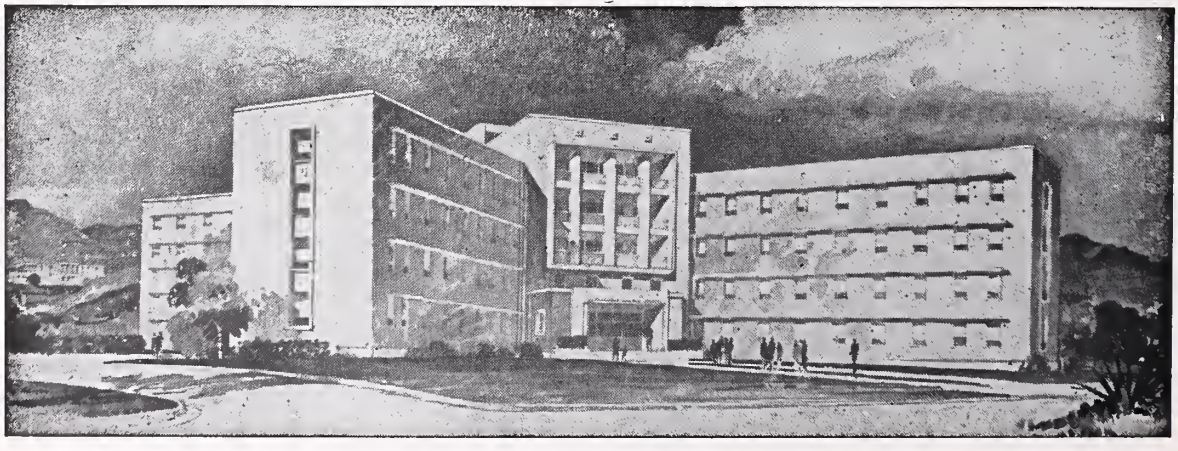
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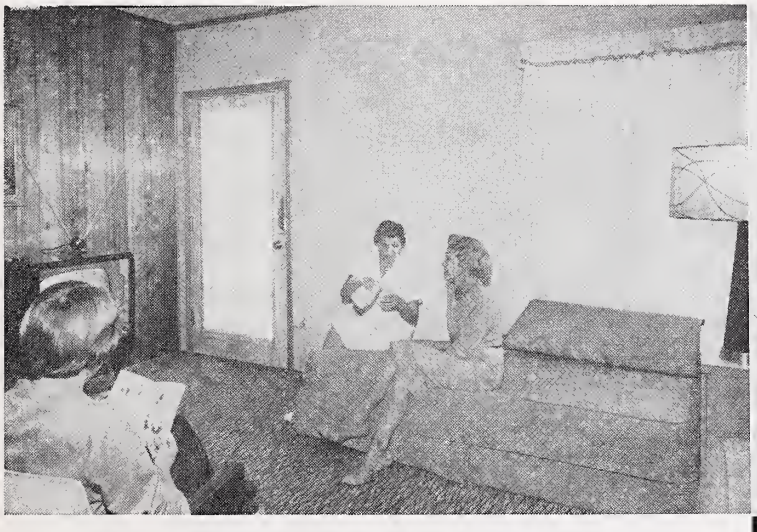
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Usual adult dosage: One or two 400 mg. tablets three times daily. Doses above 2400 mg. daily are not recommended.

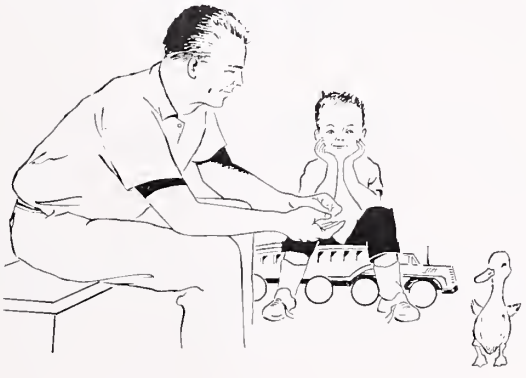
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rap•port (ră•pōrt′; rá•pôr′), n. An intimate or harmonious relation.



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Coming Meetings

19th Annual Symposium on Fundamental Cancer Research, The University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, March 4, 5, 6, 1965.

23rd Annual Meeting, U.S.-Mexico Border Public Health Assoc., Los Angeles, April 26-29, 1965.

Annual Session, Texas Medical Association, San Antonio, April 29-May 2, 1965. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Secy.

83rd Annual Meeting of the New Mexico Medical Society and 12th Biennial Meeting of the Rocky Mountain Medical Conference, La Fonda, Santa Fe, May 9-15, 1965.

Eighth Annual Ruidoso Summer Clinic, sponsored by the New Mexico Chapter of American Academy of General Practice, Ruidoso, N. M., July 19-22, 1965. Headquarters: Chaparral Motel.

62nd Annual Meeting of the Western Association of Railway Surgeons, Alameda Hotel, Avenida Juarez 50, Mexico City, October 27-30, 1965.

15th Annual Meeting of the Southwest Obstetrical and Gynecological Society, Arizona Inn, Tucson, Oct. 28-30, 1965.

47th Annual Meeting, Southwestern Medical Association, Sheraton Motor Inn, El Paso, November 4-6, 1965.



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*Roseman, E.: *Neurology* **11**:912, 1961. 33664

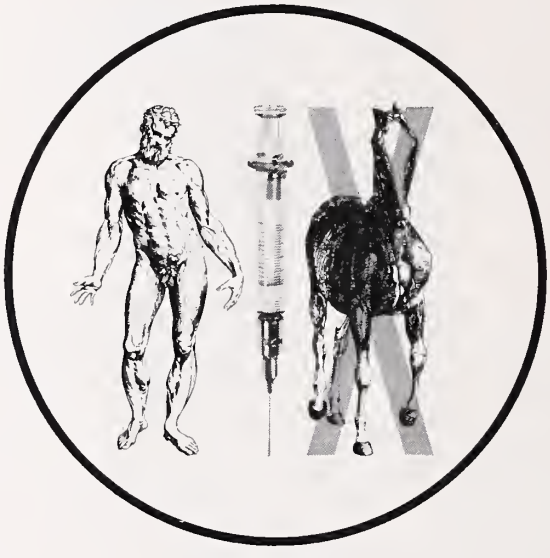
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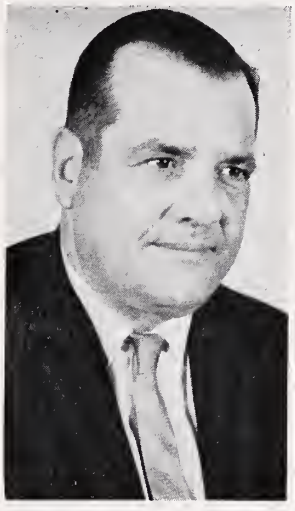
Hyper-Tet is available in 250 and 500 unit vials. A 250 unit dose is now regularly used in routine prophylactic cases. In cases where the injury is severe and where the risk of potential tetanus infection is higher, a dose in excess of 250 units may be indicated and antibiotic prophylaxis may also be advisable.[5]

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References: 1. Rubbo, S. D., and Suri, J. C.: Brit. M.J. *2*:79 (July 14) 1963. 2. Rubinstein, H. M.: Am. J. Hyg. *76*:276, 1962. 3. McComb, J. A.: New England J. Med. *270*:175 (Jan. 23) 1964. 4. Effective tetanus protective level established by Sir David Bruce. 5. Editorial: Tetanus Immunization, JAMA *161*:883, 1956.



Should We Look At Our Hole Card

SOL HEINEMANN, M.D., *El Paso*

Among the devotees of stud poker, many have the habit of taking a reassuring peek at the hole card. This usually results when one notes a change in the tenor of the play. It is both reassuring and helpful to the player who is figuring the many possibilities he faces in the opposing hands.

Now that the election has passed, I wonder how many of us have given serious thought to the possible changes that could result? There is very little doubt that President Johnson will assume he has the majority of voters and Congressmen behind his program, and from that we have heard of the "Great Society" the present trend toward increased government activities and control over the lives and economics of the nation will continue at a faster pace. This will probably affect the practice of medicine more than other business or profession which have already become partially regulated from Washington.

In considering the possible changes that will affect the medical profession, let us think of the possible effects on our future practice, overhead and taxes, and what the effects may be on our present estates.

Let's take these in order. Many of us are going to be forced to live with and under some form of government participation in the field of medicine. While we like to hear ourselves express the courage to say "I'll quit first," few of us are wealthy enough to do this; even fewer have training for other fields of endeavor. While I do not think that medicine will be socialized with a stroke of the pen, I have little faith in the defeat of some form of King-Anderson legislation in this next session of Congress. Probably under this type of legislation we will at first be busier than ever; but this in itself will lead to further regulation, in an effort to keep down the cost of medical care under this program. This will lead to several years of bickering between the profession and the government, but the profession will always lose.

Our lives, including those of our families, are certainly going to be affected by the marked inflationary trend. The cost of living will probably increase faster than income, and the cost of education for our children may be prohibitive unless we plan ahead for these increased costs. We should reconsider our insurance program, because what is adequate in 1965 may be far too little to do the job in 1970. The same should be said for Sick and Accident policies, mortgagees, etc.

While few of us have a target date for retirement, under a new system of practice many of us

may welcome the opportunity to retire. This will only be possible by careful planning. We shall also have to consider the fact that physicians will probably be forced to go under the Social Security system. While the Administration is talking about tax cuts in the field of income and excise taxes, it is in the field of the Social Security taxes that we can expect large increases for everyone in the nation. Because of this we are going to have to anticipate paying more taxes both for our employees and ourselves. Sooner or later doctors will be included under Social Security. These taxes coupled with increased overhead are going to subject us to a relative decrease in net income.

Let us now consider what we call our savings and investments to date. Since WW II it hasn't taken a genius to make good investments. While some people have done badly, the vast majority of investors have kept up with inflation. A few, using good advice, have done far better than average. While the investments were in land, stocks, or rental dwellings, most physicians have watched their savings grow and many have considered themselves astute businessmen. Under the proposed "Great Society," with increased government programs and marked changes in defense spending, with increased Social Security taxation and a more controlled economy, we are going to find that the rules have changed. It may be impossible to rely upon the friendly broker, the banker, or the president of a local industry. Many of us will need the best advice and counseling obtainable. Without this type of advice we may find that we are not keeping up with inflation, and are like the man who kept his investments in government bonds from 1940 to the present. His investments have not kept up with inflation and while he has a few more dollars available, he has lost a lot of buying power on the market today.

Let me call your attention to two articles in *Medical Economics*. The first is "Investment Counselors: Prices, Pros, Cons" by M. J. Goldberg and was printed September 28, 1959. The second article is "Where To Get the Best Investment Advice" by John Kirk and was printed August 27, 1962. These prove that good advice is available. You don't have to be a millionaire to get advice, and in the years ahead we may benefit more than those who used these services in the past.

Sure, some professionals charge one per cent annually for their services for small investors. For this they recommend what to invest in and balance your whole financial picture. Did you hesitate to pay one-half per cent plus an eight per cent loading charge on the last Mutual Fund you bought?

Let's give some thought to the future. I get the feeling Big Daddy isn't bluffing, and the stakes are too high to make the next bet just because you feel lucky, or like to see the next card in the deck.
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Treatment of Recurrent and Metastatic Carcinoma of the Breast*

Jeanne Turner Bowman, M.D., *El Paso*

Carcinoma of the breast is the leading cause of death in women 40 to 60 years of age and represents 21.7 per cent of all malignancies in females.[1] At the present time, approximately 70 per cent of these women will need palliative therapy for advanced, recurrent or metastatic breast cancer at some period in the course of their disease. It is readily apparent that this represents a problem of major magnitude in cancer management.[2]

It is the purpose of this paper to outline the palliative treatment of disseminated cancer of the breast and to present the rationale for hormonal therapy, whether achieved by irradiation, chemical or surgical means.

Control of cancers by hormonal means rests on two principles of medicine as stated by Huggins.[3] First, cancer is not necessarily autonomous and intrinsically self-perpetuating; and second, cancer can be sustained and propagated by endocrines that are functioning at normal or even subnormal levels. Those cancers retaining characteristics of the normal cells from which they arose in such a degree that the tumor cells function like the tissue of origin are, by definition, dependent tumors. These principles have led to the investigation of the therapeutic use of ablation of endocrine organs by irradiation, surgery or by medications to suppress or neutralize endogenous hormones.

The close relationship between tumor growth and hormonal balance has brought about the working hypothesis that an endogenous abnormality of steroid hormone metabolism may be responsible for the initiation and/or maintenance of tumor growth. Efforts to unravel the relationship between steroid hormones and neoplastic disease have been exerted in two principle areas: steroid hormone metabolism in diseases of those tissues that are normally involved with steroid hormone synthesis[4], and the more general problem of disordered steroid metabolism in neoplastic disease of tissue not involved in hormone biogenesis.

The main indications for hormonal control of carcinoma of the breast are: 1) advanced primary lesions beyond conventional methods of therapy, 2) as an adjunct to other established methods, particularly when the disease is widespread, and 3) as a secondary procedure for treated, but recurrent or metastatic carcinoma.

Irradiation therapy is the best method of controlling local recurrence on the chest wall and regional lymph node metastases in the axilla, supraclavicular and internal mammary regions. Surgical attempts to excise recurrences in these areas are futile and harmful.[5]

Carcinoma of the breast unfortunately metastasizes very widely. In Sproul's series of 100 autopsies in cases of carcinoma of the breast, from the Francis Delafield Hospital, there were only two cases in which metastases were not found.[5] The most frequent sites of distant metastases as found in this

*Presented at the District One Meeting of the TMA, Pecos, Texas, February 6-7, 1965.

series of autopsies are shown in Table 1.

Table 1

Most Frequent Sites Demonstrable Distant Metastases. Sproul: 100 Autopsies.

Site	Percent
Lung	69%
Liver	65%
Adrenal	49%
Kidney	17%
Spleen	17%
Pancreas	17%
Ovary	20%
Brain	22%
Thyroid	24%
Heart	11%
Diaphragm	11%
Pericardium	19%
Pleura	51%
Intestine	18%
Peritoneum	13%
Uterus	15%
Mediastinal, Peritoneal, Retroperitoneal and Inguinal Lymph Nodes	76%
Bone	71%
Skin	30%
No Metastases	2%

Radiation Therapy

Irradiation therapy finds its greatest usefulness in the palliation of the pain caused by bone metastases. Control of this symptom is one of the most important problems in carcinoma of the breast for these bone lesions often progress slowly and cause great distress. Pain may precede demonstrable radiographic changes by many months. Therefore, if the clinical evidence is suggestive of metastases, irradiation should be started at once. Radiotherapy gives definite relief from pain in approximately 75 per cent of patients with bone metastases. In some the relief lasts only a few months, but in others, it may last two or three years.

The lumbar vertebrae and pelvic bones are the most common sites of bone metastases, perhaps because in these bones the wide sinusoids of the marrow spaces are good harbors for emboli in the blood stream.

Ulceration of metastatic nodules in the skin heal with adequate radiation. The symptoms of increased mediastinal pressure from metastases in the mediastinal lymph nodes, as well as the increased pressure from supraslavicular node metastases, in most cases, can be temporarily relieved.

Irradiation is useless in some of the distant forms of metastatic carcinoma of the breast, particularly those in the abdominal viscera, brain and widespread pulmonary disease.[6] There are also limitations as to the amount of irradiation that can be applied to any one area. Radiation pneumonitis, subcutaneous fibrosis, bone necrosis and bone marrow depression are well known undesirable sequelae of irradiation applied beyond the tolerance of surrounding normal tissues.[7]

Castration by irradiation given in adequate doses effectively ablates ovarian function as shown by remission of the metastatic lesions and by reduction of estrogen excretion.

It must always be remembered that regardless of the type of irradiation used — conventional x-ray, cobalt or super-voltage — the effect is entirely local.

Hormonal Therapy

Hormonal therapy in breast malignancy is directed toward altering the endocrine environment of the tumor cell — by either additive or subtractive treatment.

The most valuable source of information concerning the use and effects of androgens and estrogens in the treatment of disseminated breast cancer are the reports of the Subcommittee on Breast and Genital Cancer of the Committee on Research of the American Medical Association. Table 2.[8]

Table 2

Hormonal Therapy

Subcommittee on Breast and Genital Cancer of the Committee on Research of A.M.A. 944 Cases. 1947-1956.

Cases	Androgen (580)	Estrogen (364)
Objective Remission, Premenopausal	20%	
Objective Remission, Postmenopausal	21%	36%
Objective Remission, 8-plus years Postmenopausal	27%	38%

The retrospective study on a series of 944 women with disseminated mammary cancer treated by androgenic hormones (580 cases) and estrogenic hormones (364 cases), from 1947 through 1956, by 60 participating investigators were analyzed and the results evaluated. The follow up, until the time of death or the date of the analysis, was

successful in 89.4 per cent (844 cases). Androgens produced objective regression in 20 per cent of premenopausal patients and in 21 per cent of postmenopausal cases. Estrogens, limited to post menopausal patients, induced regression in 36 per cent of patients. Both types of hormones were more effective after the eighth postmenopausal year, at which time both reached a plateau of performance, but with estrogens inducing a higher relative frequency of regressions (38 per cent) than did the androgens (27 per cent).

In cases with unisystemic dissemination, estrogens produced a greater degree of control in the soft tissues at all postmenopausal ages and were equal, or superior, to androgens for skeletal and visceral metastases.

The subcommittee concluded that responsive patients had significantly longer survival with androgenic and estrogenic therapy, whereas the unresponsive patients with either mode of therapy had almost identical average periods of survival comparable to untreated patients. Steroid sex hormones are sufficiently effective in naturally postmenopausal cases to deserve a primary trial in the treatment of disseminated mammary cancer. After the fourth postmenopausal year, estrogenic substances are the agents of choice.

Segaloff et al have investigated the effects of androsterone[9] and androstanediol dipropionate[10] of the clinical course and hormone excretion of women with advanced breast cancer. The results of these studies were very discouraging. Of 22 patients with advanced metastatic disease treated with androsterone, only one showed a short term objective remission. The only notable effect on the hormonal excretion pattern was the expected increase in urinary 17-ketosteroids. Of the 24 patients treated with androstanediol dipropionate, four demonstrated pronounced subjective improvement; two also showed definite objective regression of metastatic disease.

Goldenberg and Hayes[11] compared the results of testosterone propionate with a new varient compound, 2 alpha-methyl dihydrotestosterone. The new preparation was found to be as effective as testosterone in causing remissions of metastatic lesions (about 30 per cent of cases). However, there was almost no masculinizing effect from the new compound (14 per cent) as compared to the marked virilization from testosterone (57 per cent).

Unfortunately, administration of any of the known hormones has not altered the endocrine status of the patient sufficiently to produce a permanent control of the disease once metastases have become evident.[12]

Surgery

The hormonal relationship between the breast and the ovaries has been appreciated since 1896 when Thomas Beatson, a Glasgow surgeon, performed the first oophorectomies on patients with advanced carcinoma of the breast, and reported regression of the lesions for a period of time postoperatively. Modern endrocine therapy of mammary cancer began in 1951 when Huggins[13] proved that certain cancers of the breast would undergo regression following removal of the ovaries and the adrenal glands. He postulated that tumor regression was obtained by the elimination of the patient's estrogen production.

Despite the numerous controlled studies, no practical means of selecting patients for oophorectomy has been found. Neither the histological picture, nor any clinical manifestation of the disease has been helpful in predicting a favorable response.

Block et al[14] studied the effects of operative or irradiation castration in 63 patients with advanced mammary cancer. Table 3. Their conclusions were as follows:

1. Ablation therapy is indicated only when there is evidence of an active progressive disease that is no longer amenable to local resection or irradiation of a primary, recurrent or metastatic lesion.

2. Castration offered remission in approximately 35 per cent.

3. Castration gives remissions in approximately 40 per cent of the premenopausal patients, and in 22 per cent of the selected postmenopausal patients.

4. Castration therapy, when successful, gives remissions for an average of at least 10 months and individual remissions may extend several years.

5. Irradiation castration given in adequate doses effectively ablates ovarian function as shown by remissions and by reduction of estrogen excretion.

6. Postmenopausal patients who show significant estrogen excretion or who have strongly positive Mack stains are candidates for castration.

7. Castration therapy is not indicated for patients who show low levels of estrogen excretion.

8. In this series, all patients who benefited from castration showed a high precastration estrogen excretion that decreased to insigificant levels following castration.

9. Response to castration is a reliable means of predicting the hormonal sensitivity of the tumor in question.

Table 3

Effects of Operative or Irradiation Castration.
Block et al. 63 Cases.

	Remission 10-plus Months
Whole Series	35%
Premenopausal	40%
Selected Postmenopausal	22%

Originally bilateral adrenalectomy and oophorectomy were used only as palliative measures in far-advanced breast cancer. Recently, these procedures have been used as part of the primary treatment in conjunction with radical mastectomy on the basis that distant metastases are present although not demonstrable by present day methods.[15]

It has been observed that the condition of the patients with carcinoma of the breast and widespread metastases, who have secured palliation from removal of the ovaries and adrenal glands, deteriorate quite rapidly when growth in the dormant metastases is resumed. Apparently mutant malignant cells have appeared that can grow rapidly in the absence of the hormones of these glands. Therefore, acting on the concept that the chance for mutants to develop would be much greater when large masses of cancer cells are actively dividing than when metastases are of microscopic size, prophylactic bilateral adrenalectomy and oophorectomy are being performed almost immediately following radical mastectomy.

This new concept does not at present alter the fact that bilateral adrenalectomy-oophorectomy will still play a major part in the treatment of widespread metastatic carcinoma of the breast.

Like all other methods that are available for control of metastatic breast cancer, combined adrenalectomy and oophorectomy has been subjected to two major criticisms. First, it is difficult to determine beforehand which patient will benefit from the operation. Since it is obvious that approximately 50 per cent of patients derive no benefit from this major procedure, a means of preselection of endocrine-dependent cancers would be of great value. It has been shown that patients with slow-growing tumors with a long interval between onset of primary disease and the onset of metastases tend to have the best results from adrenalectomy or, in fact, from other modes of therapy as well. Experience would also indicate that there is a better chance of remission if the patient is between 40 and 57 years of age at the time of onset of carcinoma of the breast. Patients with well-differentiated breast cancer tend to have a higher incidence of response to adrenalectomy. While, in general, it may be said that the younger the patient, the less likely is the chance of a good response, numerous instances of objective and subjective response to adrenalectomy in women below the age of 40 have been recorded. A general impression, not statistically confirmed, is that patients with extensive hepatic or central nervous-system metastases have significantly less chance of getting an objective response. It is also clear that extensive pulmonary or pleural metastatic disease significantly increases the operative risk to the patient.

The second and also valid criticism of all control procedures for metastatic breast cancer is the question of long-term remission. None of the present-day methods of controlling metastatic disease have been in use long enough for a prediction of the long-term results.

Nelsen at al[16] reported 24 cases of metastatic breast cancer who underwent bilateral adrenalectomy and oophorectomy with a follow-up of three to eight years. Fifty-eight per cent of the patients obtained a significant objective response; 33 per cent of the patients lived three years or longer following surgery; 29 per cent of the patients were alive at the time of the report with an average survival of 60 months. Twenty per cent of the patients had been asymptomatic since adrenalectomy-oophorectomy with an average survival of 66 months. Table 4.

Table 4

Bilateral Adrenalectomy and Oophorectomy. 24 Cases. Nelson et al.

	Percent
Objective Remission	58%
Alive 3-plus years post-operative	33%
Alive 5 years post-operative	29%
Alive, asymptomatic 5.5 years post-operative	20%

Wilson et al[17] have observed severe exacerbations of cancer of the breast after bilateral adrenalectomy-oophorectomy in four cases. These authors postulated that this was probably due to the release of pituitary activity, previously held in check by estrogen. They concluded that women in the menopausal period were most vulnerable to exacerbations of their disease as a result of oophorectomy and/or adrenalectomy.

The value of hypophysectomy in the treatment of patients with advanced cancer of the breast has been well established. There is not, however, general agreement about which patients are most likely to respond, and many who undergo operation do not benefit from it.

Many factors appear to influence the results in different series of cases: the age of the patient at the time of operation, the relation to menopause, the total duration of the disease, the histological structure of the tumor, the sites of the metastatic lesions, and the response of the metastases to previous endocrine therapy. The most discriminating so far reported is a function, calculated from the levels of 17-hydrocorticoids and aetiocholanolone in the urine, which shows a close correlation with the clinical result.

McCalister et al[18] have reported an analysis of three principal factors influencing the response to hypophysectomy. These factors were: 1) the sites of the lesions, 2) the relation to menopause, 3) the time at which the operation was performed. These factors were analyzed in 75 consecutive patients who survived the operation. Table 5. These

Table 5

Effects of Hypophysectomy. 75 Cases. McCalister et al.

	Cases	Percent
Whole Series	32/75	43%
With Osseous Metastases	27/52	52%
Without Osseous Metastases	5/23	22%
Premenopausal	13/21	62%
Postmenopausal	19/54	35%
Surgery: Less than 6 months	32/45	51%
Surgery: More than 6 months	9/30	30%
Premenopausal with Osseous Metastases	13/17	75%

authors found that previous endocrine therapy influenced the response in various ways. The age of the patient, the total duration of the disease, and the histological structure of the tumor did not influence the result.

It was found that patients who had bony metatases, with or without active disease in the region of the primary growth, had a higher remission rate than those whose metastases were limited to distant viscera and soft tissues. Remissions occurred in a higher proportion of patients who were still menstruating than those who had had natural menopause. The response was apt to be more favorable when hypophysectomy was performed within six months of the disease becoming uncontrolled than when it was delayed beyond this period. The best results were obtained in premenopausal women whose disease had metastasized to bone and were operated on within six months of this event.

The above figures differ from those of Ray and Pearson[19] in that they fail to show any difference in responsiveness between metastatic tumors which were slowly and those which were rapidly progressing. The period betwen mastectomy and the appearance of metastases, the "free interval," in carcinoma of the breast varies from less than one to 25 years. Ray and Pearson found that when this free interval was less than a year, the remission rate was 33 per cent; between one and two years, it was 47 per cent; and between two and four years, 52 per cent; and when over four years it reached 66 per cent. This remission rate is of obvious value in the selection of patients likely to respond to hypophysectomy.

Boesen et al[20], another group of British workers, have reported on 111 unselected patients with disseminated carcinoma of the breast. The obtained regression or arrest in 42 per cent. They concluded that a favorable response is more likely in patients who had responded well to previous endocrine therapy and in those who had had a long "free interval".

A preliminary evaluation of the results of adrenalectomy and hypophysectomy for the palliation of metastatic mammary carcinoma at 12 centers has been made by the Joint Committee on Endocrine Ablation Procedures in Disseminated

Mammary Carcinoma.[21] Objective response or regression of metastatic lesions was noted after adrenalectomy in 31.7 per cent of patients (100/315) and in 31.2 per cent following hypophysectomy (112/358). For responsive cases, survival averaged 20.6 months with adrenalectomy; for nonresponders, the comparable figures were 6.5 and 7.0 months.

Chemotherapy

Ultimately far advanced carcinoma of the breast will cease to respond to the so-called conventional therapy, and recourse can be made to the alkylating agents, colchicine, antimetabolites and radioactive isotopes. Most of these studies have met with disappointing results.

Bateman and Carlton[22] reported a series of 286 patients with cancer of the breast who were treated with Thio-TEPA by every known route. About two-thirds of the patients showed some objective improvement lasting about eight months. This form of treatment has been used with some success in liver metastases and where pleural and peritoneal effusions are present. Table 6. Watson

Table 6

Effects of Thio-TEPA. Bateman and Carlton. 287 Cases.

	Percent	Duration
Objective Remission	66%	8 months
Watson and Turner. 34 Cases.		
Effects of Thio-TEPA and Testosterone		
Objective Remission	88% (30/34)	

and Turner[23] reported a series of 34 cases in which they combined the use of Thio-TEPA with testosterone. They obtained an 88 per cent improvement (30/34).

Encouraging results have been obtained by Storaaski et al[24] in the treatment of osseous metastases with radioactive phosphorous alone and in combination with adrenalectomy. Table 7. Sixty-

Table 7

Effect of P^{32} and/or Adrenalectomy. 64 Cases with Osseous Mestastases. Storaaski et al.

	Objective Remission	Cases
P^{32} alone	26%	42
P^{32} with Adrenalectomy	68%	22
Adrenalectomy Alone	40%	

four patients with extensive osseous metastases from carcinoma of the breast were treated with radioactive phosphorous. Forty-two cases were treated with P^{32} alone and objective benefit was obtained in 26 per cent. Twenty-two patients were treated with bilateral adrenalectomy and P^{32} and objective benefit was obtained in 68 per cent. They concluded that patients in negative calcium and phosphorous balance with predominantly osseous metastases which are known to be hormonally dependent have the greatest chance of responding to bilateral adrenalectomy and P^{32}.

The fluorinated pyrimidine, 5-Fluorouracil, has been given to a number of patients with advanced carcinoma of the breast. Definite objective regression of skin nodules as well as liver metastases have been observed. However, the toxicity of the compound, and the rapid growth of tumors which initially regressed are two of the main disadvantages of the drug. Tumor regrowth requires the administration of repeated courses of the drug, and the toxicities require frequent laboratory and clinical evaluation. There is no doubt that the drug has a place in the management of advanced cancer, but it is relatively narrow and as yet not well established.[25]

Summary

The management and treatment of patients with advanced carcinoma of the breast have been presented. Much can be offered to these patients and it is most important that the patients and her family should not be allowed to feel that the physician has given up hope. In deciding between the ablation procedures and the administration of hormones or chemotherapy, one must temper enthusiasm with clinical judgment and consider the patient's family and financial situation before involving them in the heavy costs of hospitalization followed by complicated and costly substitution therapy, when no cure can honestly be promised. Nothing is lost by a trial of conservative therapy, which can be conducted at home or as a hospital out-patient under the supervision of the family physician or the radiotherapist. In the words of Dr. Trudeau, our duties regarding the practice of medicine are, "To cure sometimes, to relieve often, and to comfort and support always"[26].

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Railway Surgeons Elect Officers

Dr. Ivan Ingram of San Francisco was elected President of the Western Association of Railway Surgeons at the 61st annual meeting of the organization in Sun Valley, Idaho.

Other new officers are Dr. Samuel E. Senor, St. Joseph, Mo., First Vice-President; Dr. John R. Ong, Jr., Dallas, Second Vice-President; Dr. Harry O. Hund, San Rafael, California, Treasurer; Dr. Graham Owens, Kansas City, Missouri, Secretary; and Dr. John C. Mitchell, Salina, Kansas, Chairman of the Executive Committee.

The 1965 meeting will be held in Mexico City, Oct. 27-30 at the Alameda Hotel, Avenida Juarez 50.

Speakers at scientific sessions were Dr. George T. Manilla, Elko, Nevada; L. D. Clark, Omaha, District Claim Agent for the Union Pacific; Dr. Linares B. Johnson, San Francisco; Maynard E. Parks, St. Louis, Vice-President of Personnel for the Missouri Pacific Railroad; Dr. Richard E. Ferguson, San Francisco; Dr. Roland S. Kieffer, Medical Director of the Missouri, Kansas and Texas Railroad; Dr. Maire McAuliffe, San Francisco; Dr. Robert W. Weber, Salina, Kansas, Assistant Clinical Professor of Medicine at the University of Kansas School of Medicine; Dr. Donald F. Harvey, San Francisco, F. B. Lewis, Omaha, Manager of Safety and Courtesy for the Union Pacific Railroad, Dr. John R. Crew, San Francisco; J. E. Schwartz, Chief of the Division of Disability Benefits of the U. S. Railroad Retirement Board; and Dr. Benjamin Rosen, Chief Medical Officer, Division of Disability Benefits, Bureau of Retirement Claims, U. S. Railroad Retirement Board.

In Memoriam
Dr. Louis G. Jekel



Dr. Louis G. Jekel, 56, a pioneer in Dermatology in Arizona and a prominent and beloved physician of the Southwest, died in Phoenix November 18, 1964, of a chronic heart ailment. His death is a great loss to the medical profession of the Southwest, and certainly no one can replace him in the hearts of the many close friends he made during his years of practice in Arizona.

Dr. Jekel received a B.S. and M.D. degree from Washington University in St. Louis and did graduate work in Internal Medicine and Dermatology at Barnard Skin and Cancer Hospital from 1934 to 1938. He then located in Phoenix, where he practiced Dermatology until his death. Though a specialist in Dermatology, he always maintained an interest in medicine in general, was active in medical circles and was a member of many medical societies, not only those in the Southwest, but of other regional and national organizations.

Dr. Jekel was a former President of the Southwestern Medical Association and the Southwestern Dermatological Society and was instrumental in organizing the second. He was a diplomate of the American Board of Dermatology; a Fellow of both the American Academy of Dermatology and the American College of Physicians; a member of the Southern Medical Association; the Society of Investigative Dermatology; as well as all his local, state and national societies. He was an active member and former Vice-President of the Pacific Dermatological Society.

Surviving Dr. Jekel are his wife, Margaret, of Phoenix, two daughters, Judith of Long Beach, California, Mrs. Charles Barrett of Tempe, Arizona, a son, Louis, Jr., of Phoenix, two brothers, a sister, and a grandson.

Louie Jekel was a kindly gentlemen, a dedicated physician, a well informed person, a most hospitable host, and a man of considerable charm and wit. To those of us who have been fortunate enough to be close friends with Louie and Margaret during a good many years, Louie's death is a great personal loss, and he will be sorely missed at all meetings.

— Leslie M. Smith, M.D., El Paso

Announce Speakers For N. M. Medical Society Annual Meeting

Several speakers have been announced for the combined meeting of the Rocky Mountain Medical Conference and the New Mexico Medical Society in Santa Fe, May 9-15, 1965.

Speakers include:

Dr. Edward J. Stainbrook, Professor of Psychiatry, University of Southern California School of Medicine.

Dr. Hollis G. Boren, New York, Director of the Trudeau Foundation.

Dr. William D. Cochran, Boston.

Dr. Oscar Auerbach, Senior Medical Investigator of the Veteran's Administration Hospital, East Orange, N. J.

Dr. Audrey Brown, Associate Professor of Pediatrics, University of Virginia School of Medicine.

Dr. Brown N. Dobyns, Professor of Surgery, Western Reserve School of Medicine, Cleveland.

Dr. Alfred Bollet, Associate Professor of Medicine, University of Virginia School of Medicine.

Dr. Norman Shumway, Stanford University School of Medicine.

Dr. Keith Reemstma, Tulane University School of Medicine.

The clinical program will start May 12. The Council will meet at 2 p.m. May 9, the House of Delegates at 2 p.m. May 10 and 2:30 p.m. and 7:30 p.m. May 11. The Orientation Course is scheduled for 8:30 a.m. May 11. Headquarters for the joint meeting will be the La Fonda Hotel.

New Mexico Officials Discuss Health Care

Health Care—Present and Future was the subject for a one-day conference of County Medical Officers in Santa Fe, January 30, at a meeting sponsored by the Public Relations Committee of the New Mexico Medical Society.

Speakers in a symposium on "Medical Care in New Mexico" were LaVon Burrell, Albuquerque, Director of the New Mexico Blue Cross—Blue Shield, who discussed "Voluntary Health Insurance in New Mexico—its Growth, Past, Present and Future;" James Black, Albuquerque, Chairman of the New Mexico Health Insurance Council, "Commercial Health Insurance in New Mexico—Its Growth, Past, Present and Future"; Dr. Reginald H. Fitz, Dean of the University of New Mexico Medical School, "Physician Supply in New Mexico—Past, Present and Future"; Dr. Robert L. Zobel, Albuquerque, Indian Health Area Director, "Health Services for Indians—Past, Present and Future"; and Leo Murphy, Santa Fe, Director of the New Mexico Department of Public Welfare, "Health Services of Department of Public Welfare—Past, Present and Future".

Other speakers and their subjects were Dr. Harry Ellis, Santa Fe, chairman of the State Society's Legislative and Public Policy Committee, "The Medical Society's Legislative Program"; Dr. Robert P. Beaudette, Raton, President-Elect, New Mexico Medical Society, "The AMA's National Advertising Program"; Dr. Thomas L. Carr, Albuquerque, Vice-President, New Mexico Medical Society, "Report on AMA's National Conference on Kerr-Mills"; and Mrs. Roy Goddard, Albuquerque, President of the State Society's Auxiliary, "The Auxiliary's Role in Assisting the Medical Society".

El Paso OB & Gyn Society Sponsors Meeting

Outstanding guest speakers in the field of Obstetrics and Gynecology participated in a meeting in El Paso, January 23, held in conjunction with a joint dinner of the El Paso County Medical Society and the R. E. Thomason General Hospital Staff, and with the annual conference of the Texas Medical Association's Committee on Maternal Mortality.

Guest speakers were Dr. Jack A. Pritchard, Professor and Chairman of the Department of Obstetrics and Gynecology at the University of Texas Southwestern Medical School, Dr. David L. Barclay, Associate Professor of OB and Gyn at Tulane School of Medicine, Dr. Robert A. Johnson, Head of the Department of OB Gyn at St. Joseph's Hospital in Houston, Dr. John Jewett, Boston, Head of the Maternal Mortality Committee in Boston, Dr. Steve Schanzer, M.C., Chief of OB and Gyn at William Beaumont General Hospital in El Paso, Dr. Homer Jacobs, El Paso, and Dr. Werner E. Spier, El Paso. Dr. James R. Morgan was chairman of the meeting, which was sponsored by the El Paso Obstetrical and Gynecological Society and held at Thomason General Hospital. Subjects were "Vena Cava Ligation", "Reflections of An Obstetrician", "Hypofibrinogenemia", "An Analysis of Maternal Deaths from the Boston Area" and "Sepsis".

Dr. Pritchard spoke on "Is Pregnancy a Disease?" in the Second Annual Dr. Nathan Kleban Memorial Lecture at the dinner meeting, held January 22. Dr. Kleban, prominent El Paso internist and President of District One of the Texas Medical Association, died in 1963.

Southwest Blood Banks Elects Trustees

Dr. D. W. Melick of Phoenix has been elected Chairman of the Board of Trustees of Southwest Blood Banks, Inc., at its annual meeting in Scottsdale, Arizona.

Other members of the Board of Trustees elected at the meeting are Dr. Maynard S. Hart, El Paso, who was re-elected Vice-Chairman; Dr. T. L. Carr, Albuquerque, Secretary; Dr. Irving W. Moody, Houston, Treasurer; and Dr. Zeph B. Campbell, Phoenix, member of the Board.

Southwest Blood Banks was established in Phoenix in 1943, now operates 18 blood banks in 10 states, a wholly-owned subsidiary, Blood Service Plan Insurance Company, and is the nation's largest medically-sponsored, self-supporting, not-for-profit blood banking system.

Biomedical Sciences Instrumentation Symposium

The Third National ISA (Instrument Society of America) Biomedical Sciences Instrumentation Symposium will be held in Dallas, April 19-21, 1965, and will have as its theme "The Transduction, Transformation and Display of Physiological Variables".

Among the speakers will be Dr. A. R. Kahn, Director of Physiological Research for Beckman Instruments, Inc., Dr. J. R. Cox, Director of the Biomedical Computer Laboratory at Washington University, Dr. R. T. Kado of The Brain Research Institute of the University of California at Los Angeles, and Dr. L. V. Berkner, President of the Graduate Research Center of the Southwest in Dallas. Headquarters for the meeting will be the Statler Hilton.

Plans Progress For Southwestern Medical Ass'n. Meeting, El Paso, Nov. 4-6

The 47th Annual Meeting of the Southwestern Medical Association will be held in El Paso, November 4-6, 1965, according to an announcement by Dr. Clement C. Boehler, El Paso, President of the Association.

Dr. Boehler has named Dr. Gordon L. Black, General Program Chairman, Dr. A. Robert Nering, Scientific Program Chairman, and Dr. Sol Heinemann, Secretary-Treasurer, Chairman of Exhibits.

Headquarters for the meeting will be at the Sheraton Motor Inn near the Sun Bowl.

A total of 156 physicians registered for the 1964 meeting at Las Vegas, Nevada, where the scientific program was presented by the School of Medicine at the University of California, San Francisco Medical Center.

In addition to Dr. Boehler and Dr. Heinemann, officers of the Association are Dr. Frank A. Rowe, Albuquerque, President-Elect; Dr. Zigmund W. Kosicki, Santa Fe, Vice-President; and members of the Executive Committee, in addition to the above physicians, Dr. Frank A. Shallenberger, Jr., Tucson, Dr. Robert F. Boverie, El Paso, Dr. Louis W. Breck, El Paso, Dr. John S. Carlson, Phoenix, Dr. Charles W. Carroll, Las Cruces, New Mexico, Dr. Homero Galindo, Juarez, Mexico, Dr.. Frederico Sotelo, Hermosillo, Sonora, Mexico, and Dr. J. Warner Webb, Jr., Tucson.

BOOK REVIEW

Atlas of General Surgery

C. V. Mosby Company, 1964

(2nd edition) by Joseph R. Wilder

This 317 page Atlas contains a descriptive text with accompanying illustrations of 105 operative procedures. These deal mainly with abdominal operations; but included are some neck, vascular, hand, and pelvic surgical procedures. The author doesn't claim the operations as being original and, except in three or four instances, doesn't present alternative techniques for performing a procedure.

The medical illustrator, Miss Shirley Baty, has executed clear, diagramatic illustrations. The Atlas would be most useful to one undergoing surgical training as a supplement to supervised technical operating room experience. To the trained surgeon, it would provide a review of the more important, though not detailed, steps in performing a specific operation.—*George W. Iwen, M.D.*

Dr. Floyd New President Bernalillo County Medical Association

Dr. Vaun Floyd, Albuquerque, has been elected President of the Bernalillo County Medical Association for the coming year. He succeeds Dr. Roy Goddard.

Other new officers are Dr. Eugene Szerlip, First Vice-President; Dr. J. Hunt Burress, Second Vice-President; and Dr. Elmo D. Anderson, Secretary-Treasurer.

Members of the Association's Board of Trustees for 1965. include Dr. O. Douglas Johnson, Dr. Joseph Gordon, Dr. C. M. Kemper, Dr. E. H. Wood, Dr. Edwin B. Herring, Dr. John F. Griffin, and Dr. John J. Corcoran.

Scott — White Conference

Guest speakers for the scientific program of the 13th Annual Scott and White Conference in Medicine and Surgery at Temple, Texas, February 21-23, 1965, will be Dr. Malcolm B. Dockerty, Head of the Section of Surgical Pathology, Mayo Clinic, and Dr. Andre J. Bruwer, Radiologist at the Tucson Medical Center in Tucson. The after-dinner speaker will be Attorney General Waggoner Carr of Texas.

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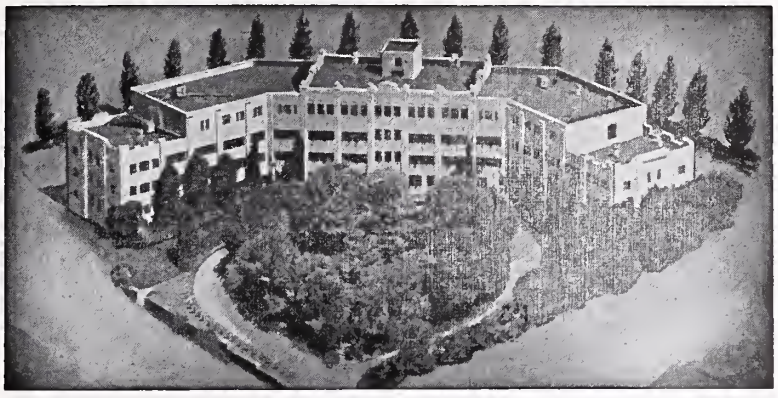
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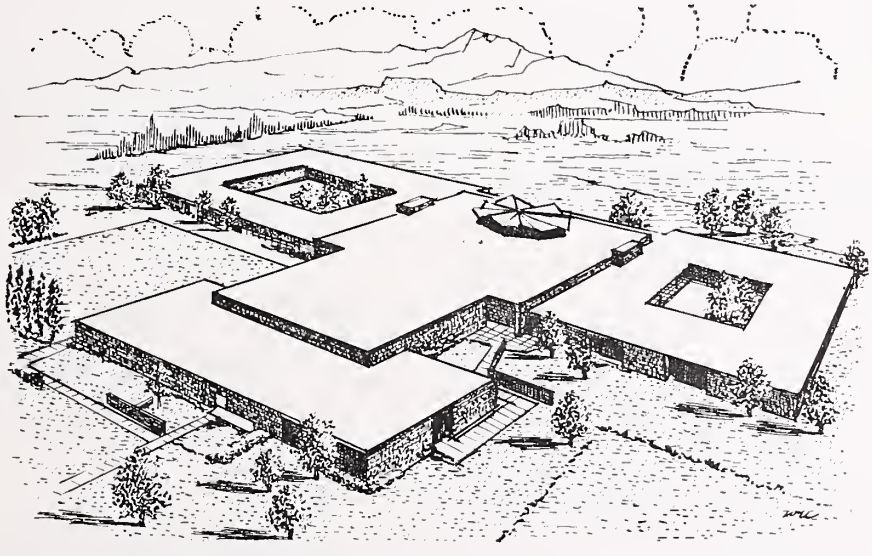
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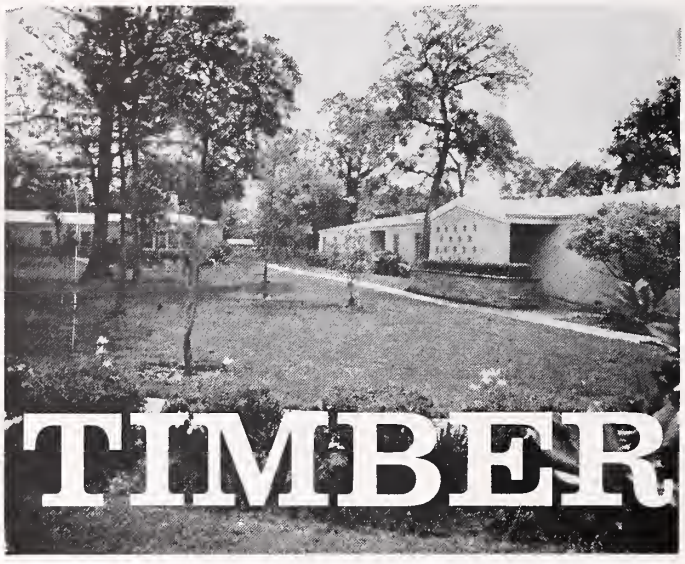


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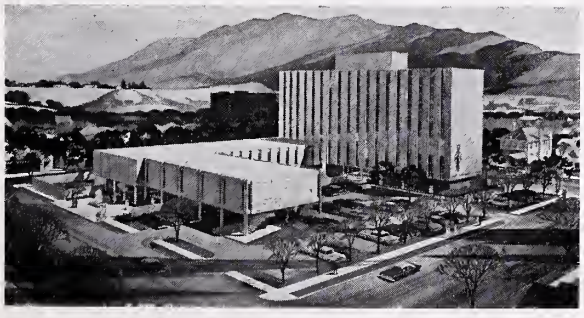
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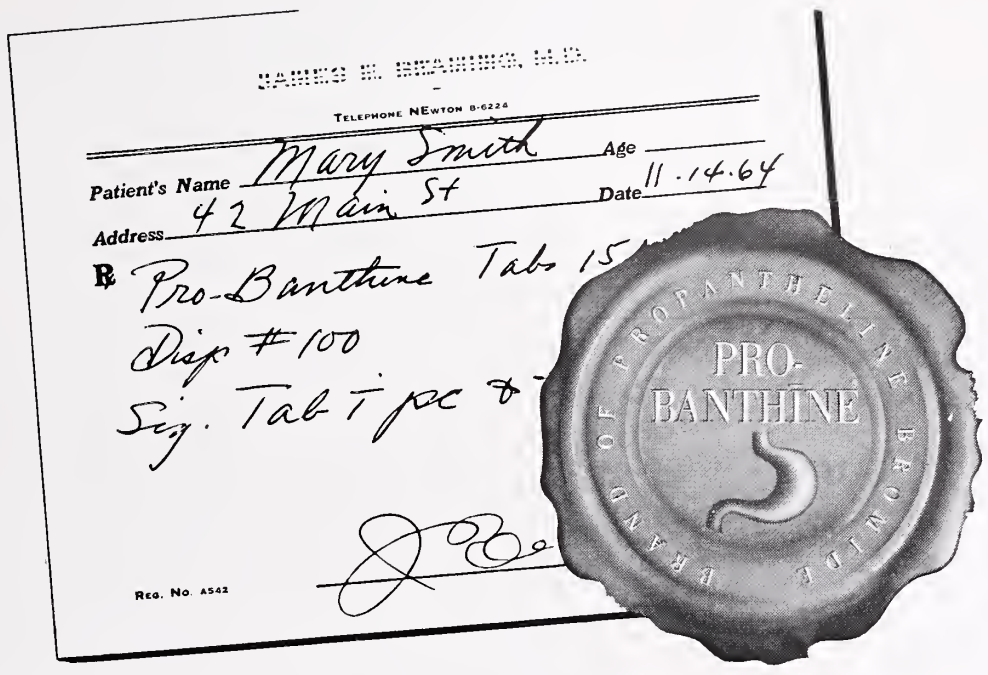
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(propantheline bromide)

Historically, reduction of acid and motility in peptic ulcer has been approached through the use of antacids, dietary management and surgery.

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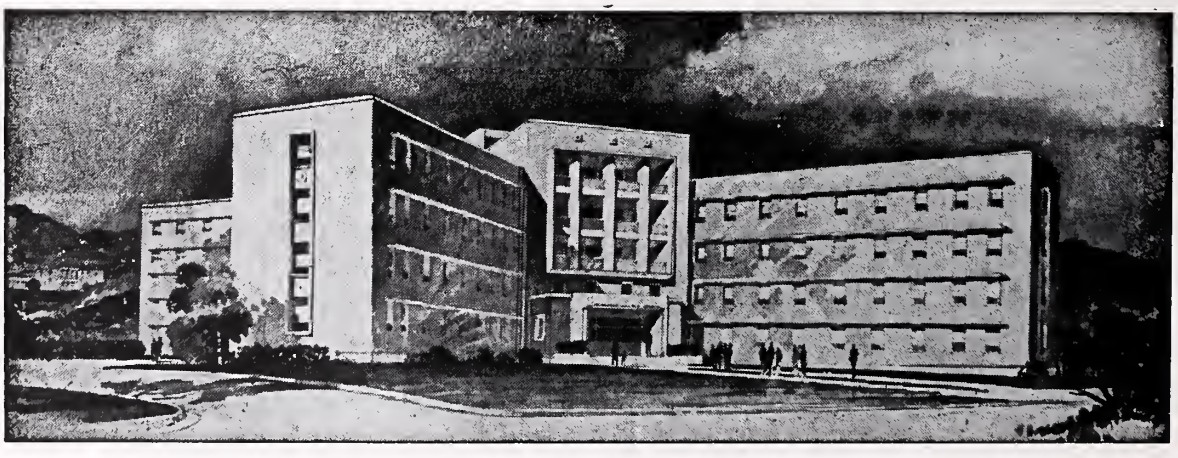
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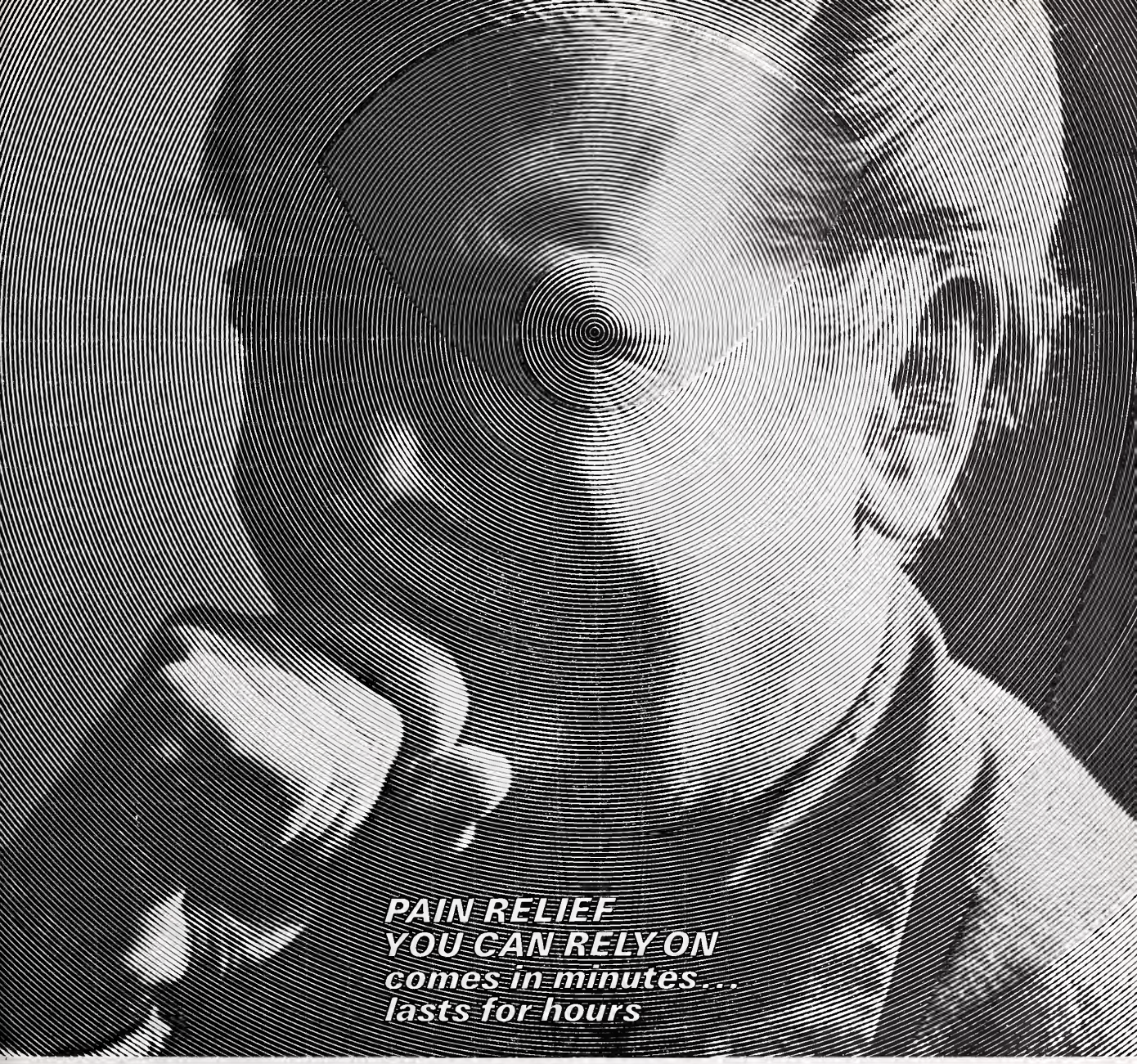
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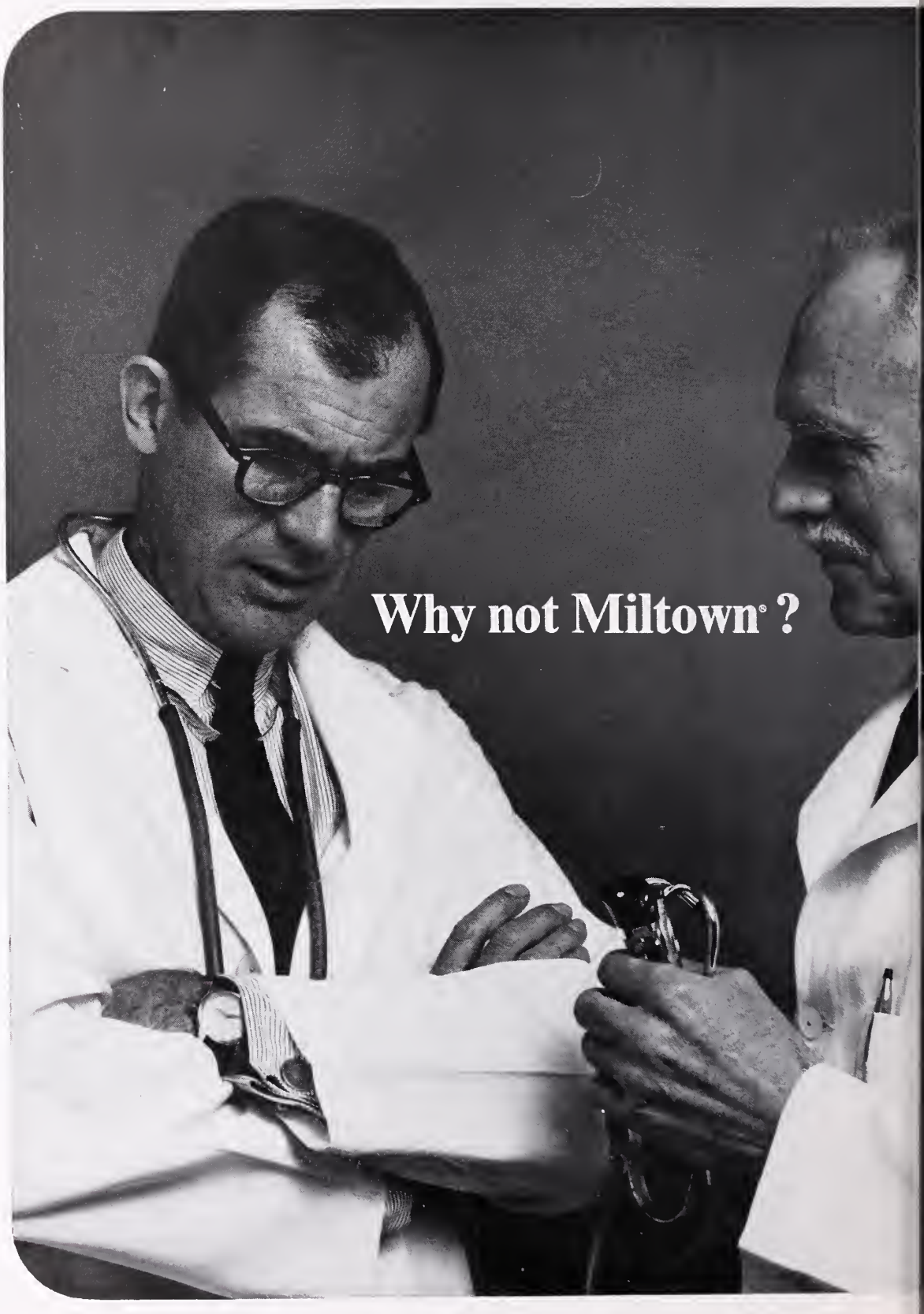
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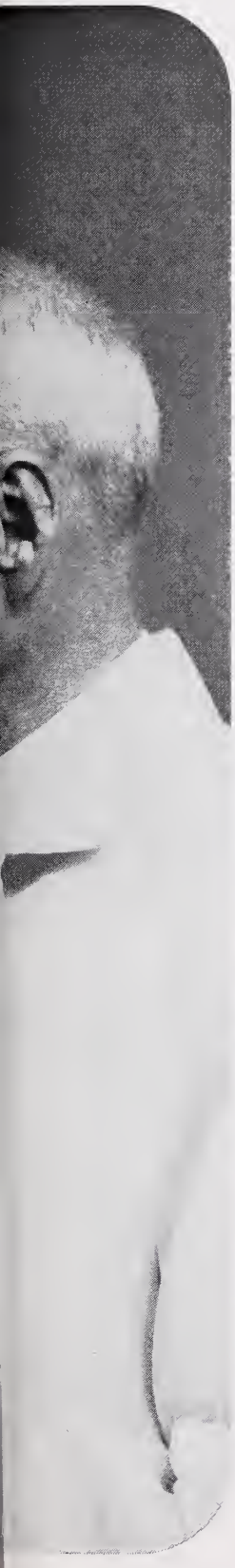
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Side effects: Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

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Supplied: In two strengths: 400 mg. scored tablets and 200 mg. coated tablets.

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Approximately 25% of American women bearing a first child are less than 20 years old. In 1960, teen-age girls gave birth to 593,746 children in the United States.

Many surveys indicate that teen-age girls are the most poorly nourished group in American society. Those who start their families in the early and mid-teens are most apt to be ill equipped to meet the combined nutritional burdens of pregnancy, lactation and adolescence.

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Lactation greatly increases maternal requirements for calories, proteins, minerals, and vitamins. The lactating mother supplies a 4-week old infant with an estimated 6.2 gm. protein, 42 gm. lactose, 182 mg. calcium, 0.6 mg. iron, and 20-30 mg. ascorbic acid daily.

A well-balanced varied diet adjusted in quantity to satisfy simultaneously the needs of pregnancy and adolescence offers the best nutritional assurance for a healthy teen-age mother and child. Basic to this diet is a milk quota of at least 1 quart a day during pregnancy and 1½ quarts a day during lactation—or their equivalent in other dairy products.



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Side Effects: Occasional nausea, vomiting and diarrhea. **Precautions:** With any broad spectrum antibiotic, the patient should be carefully watched for signs of secondary infections caused by nonsusceptible organisms. Use of tetracycline drugs, particularly long-term use, during periods of tooth development may cause discoloration of teeth. Particular caution should be observed if renal impairment exists.

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1. Clinical report on file, Medical Department, A. H. Robins Co., Inc.
2. Schiller, I. W. and Lowell, F. C.: New England J. Med. 261:478, 1959.

BRIEF SUMMARY: Indications: Dimetapp reduces nasal secretions, congestion, and postnasal drip for symptomatic relief of colds, U.R.I., sinusitis, and rhinitis. **Contraindications:** Antihistamine sensitivity. Not recommended for use during pregnancy. **Precautions:** Administer with caution in the presence of cardiac or peripheral vascular diseases and hypertension. Until the patient's response has been determined, he should be cautioned against engaging in operations requiring alertness. **Side Effects:** Hypersensitivity reactions including skin rashes, urticaria, hypotension and thrombocytopenia have been reported on rare occasions. Drowsiness, lassitude, nausea, giddiness, dryness of the mouth, mydriasis, increased irritability or excitement may be encountered.

See product literature for complete prescribing information.

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Epstein[1] found glycopyrrolate's intensive antisecretory action to be exemplary. Breidenbach[2] was impressed by its pronounced antispasmodic effects. And Young and Sun[3] reported that a 2 mg. oral dose "did not affect gastric emptying or intestinal transit time. . . ." According to Slanger[4], the absence of annoying side effects is an important plus factor ". . . for it permits ready individualization of dosage for control of mild to severe symptoms." Posey[5] sums it up when he says, "In effect, with glycopyrrolate, one approaches an ideal agent for the management of peptic ulcer. With it a vagolytic effect may be obtained without interfering with gastric emptying—the medical equivalent to vagectomy plus an adequate drainage procedure."

We invite you to try Robinul (glycopyrrolate) in your practice. Discover firsthand how several subtle advantages add up to make it the superior anticholinergic agent.

BRIEF SUMMARY

INDICATIONS: In addition to its primary indications for duodenal and gastric ulcer, glycopyrrolate is indicated for other G-I conditions which may benefit from anticholinergic therapy. Robinul-PH Forte (glycopyrrolate 2 mg. with phenobarbital) is indicated when these situations are complicated by mild anxiety and tension.

CONTRAINDICATIONS: Glaucoma, urinary bladder neck obstruction, pyloric obstruction, stenosis with significant gastric retention, prostatic hypertrophy, duodenal obstruction, cardiospasm (megaesophagus), and achalasia of the esophagus, and in the case of Robinul-PH Forte, sensitivity to phenobarbital.

PRECAUTIONS: Administer with caution in the presence of incipient glaucoma.

SIDE EFFECTS: Dryness of the mouth, blurred vision, urinary difficulties, and constipation are rarely troublesome and may generally be controlled by reduction of dosage. Other side effects associated with the use of anticholinergic drugs include tachycardia, palpitation, dilatation of the pupil, increased ocular tension, weakness, nausea, vomiting, headache, dizziness, drowsiness, and rash.

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phenobarbital 16.2 mg.
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REFERENCES: 1. Epstein, J. H.: Am. J. of Gastroent., 37:295, March, 1962. 2. Breidenbach, W. C.: Investigative Clinical Report, March, 1961. 3. Young, R., and Sun, D. C. H.: Ann. N. Y. Acad. Sc., 99:174, Feb., 1962. 4 Slanger, A.: Journal of New Drugs, 2:215, July-Aug., 1962. 5. Posey, E. L., Jr.: Am. J. Dig. Dis., 7:863, Oct., 1962.

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Hyper-Tet is a gamma globulin fraction of venous blood from hyperimmunized humans and contains no heterologous protein. Far fewer units of Hyper-Tet are required for prophylaxis than with equine or bovine antitoxin. Recent studies[1-3] show these lower dosages of tetanus immune globulin (human) established protective levels well above the recommended immunity level[4] for up to 5 weeks.

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The tetanus antitoxin without horse serum and its reactions

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Hyper-Tet is available in 250 and 500 unit vials. A 250 unit dose is now regularly used in routine prophylactic cases. In cases where the injury is severe and where the risk of potential tetanus infection is higher, a dose in excess of 250 units may be indicated and antibiotic prophylaxis may also be advisable.[5]

Side Effects and Precautions: The likelihood of anaphylactoid or serum reactions due to intramuscular injection of gamma globulin is remote. Very rare serious reactions have been reported, however, and their extreme rarity makes it impossible to predict their occurrence. Slight soreness at and over the injection site may be noted. Do not give intravenously. There are no known contraindications.

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CUTTER *Laboratories*
Berkeley 10, California

References: 1. Rubbo, S. D., and Suri, J. C.: Brit. M.J. *2*:79 (July 14) 1963. 2. Rubinstein, H. M.: Am. J. Hyg. *76*:276, 1962. 3. McComb, J. A.: New England J. Med. *270*:175 (Jan. 23) 1964. 4. Effective tetanus protective level established by Sir David Bruce. 6. Editorial: Tetanus Immunization, JAMA *161*:883, 1956.



Dr. Budwig Elected President TMA District One

Dr. Ira A. Budwig, Jr., El Paso, was elected President of District One of the Texas Medical Association at its annual meeting in Pecos, February 6 and 7, 1965.

Other new officers are Dr. Cecil Robinson, Kermit, President-Elect, and Dr. Mario Palafox, El Paso, Secretary-Treasurer. Dr. George Hoffman, Fort Stockton, was the retiring President. Dr. William R. Gaddis, El Paso, retiring Secretary-Treasurer, was General Chairman of the meeting, and Dr. J. Leighton Green, El Paso, was in charge of the postgraduate program on February 7, which was sponsored by the University of Texas Graduate School of Biomedical Sciences.

Speakers at the meeting were Dr. Max E. Johnson, San Antonio, TMA President; Dr. Robert S. Nelson and Dr. John A. Shively, both of The University of Texas M.D. Anderson Hospital and Tumor Institute in Houston; Dr. Fred B. Kessler, Clinical Instructor in Surgery at the Baylor University College of Medicine; and Dr. John M. Verosky, Dr. George W. Iwen, Dr. Jeanne Turner Bowman and Dr. Dale F. Rector, all of El Paso.

Dr. Budwig was born in Chicago and received his B.S. degree from the University of Illinois and his M.D. from the University of Illinois College of Medicine. He interned and then took a two-year residency in Pediatrics at Cook County Hospital in Chicago, which was interrupted by two years of service with the Navy in the Pacific with the Medical Corps. He began the practice of medicine in El Paso in 1951 in association with the group now known as Drs. Bennett, Budwig, McNeil and Verosky.

Dr. Budwig is certified by the American Board of Pediatrics, and is a Fellow in the American Academy of Pediatrics and the American Academy of Cerebral Palsy. Dr. Budwig is a member of the TMA Rehabilitation Committee and the State Medical Advisory Board of United Cerebral Palsy of Texas. He is a former Delegate to the TMA.

He is a Board Member and Medical Director of the Cerebral Palsy and Rehabilitation Center in El Paso, posts he has held for the last six years. He is a charter member, former Chairman of the Board, and former President of the Memorial Park School for Exceptional Children in El Paso. He is Chief-of-Staff at Thomason General Hospital, former Chief-of-Staff at Hotel Dieu, and former Chief of Pediatrics at Hotel Dieu, Providence Memorial Hospital, and Thomason General Hospital. He is a member of Alpha Omega Alpha, Honorary Medical Fraternity.

He and his wife, the former Marjorie Ann Sanders, reside at 2304 Red Bluff Road with their children, Sandra 13, Andy 10, and Ralph 9.



DISTRICT ONE—Present at the annual meeting of District One of the Texas Medical Association in Pecos, February 6-7, 1965, were, front row, left to right, Mrs. George Turner, El Paso, Past President of the AMA Auxiliary, Dr. Max E. Johnson, San Antonio, TMA President, and Mrs. Russell L. Deter, El Paso, President-Elect of the TMA Auxiliary; back row, left to right, Dr. Russell Holt, El Paso, District One Councilor, Dr. Ira A. Budwig, Jr., El Paso, new President of District One, Dr. George Hoffman, Fort Stockton, retiring President, Dr. William R. Gaddis, El Paso, General Chairman for the meeting and retiring Secretary-Treasurer, and Dr. Mario Palafox, El Paso, new Secretary-Treasurer.

UCLA Medical School Will Present Program For Southwestern Meet

The University of California at Los Angeles School of Medicine will provide the Faculty for the 47th Annual Meeting of the Southwestern Medical Association in El Paso, November 4-6, 1965.

The scientific program is under the direction of Dr. A. Robert Nering of El Paso.

The general subject for the three-day meeting will be Chest Diseases. In addition, a discussion of Practical Office Psychiatry has been scheduled tentatively on the agenda.

Dr. Nering has announced that those attending the meeting will be given nine hours credit for postgraduate study by the AAGP. Dr. Charles R. Smart, Assistant Professor of Surgery and Assistant Director of Postgraduate Medical Education at the School of Medicine, is preparing details of the program. Headquarters for the meeting will be at the Sheraton Motor Inn near the Sun Bowl.

The Assessment of Cardiac Defects By Dye-Dilution Technics*

D. A. Malooly, M.D., J. M. Verosky, M.D., E. S. Crossett, M.D.,
J. A. Hancock, Ph.D., and G. W. Iwen, M.D., *El Paso*

Selection of cardiac patients for surgical treatment requires not only an accurate diagnosis of the lesion present but also an assessment of the magnitude of the hemodynamic abnormality. Cardiac catheterization in conjunction with a good clinical workup, electrocardiography, vectorcardiography, phonocardiography, conventional radiography, and angiocardiography is used in this assessment. An important part of this multi-faceted approach involves the use of dye-dilution technics[1] within the framework of cardiac catheterization. Certain of these technics used in the cardiac catheterization laboratory at Providence Memorial Hospital will be discussed with their application to the selection of patients for cardiac surgery.

Method

A Cournand needle is inserted into the right brachial artery for blood sampling through a XC-50B Wood cuvette oximeter. A 50 ml. lubricated heparinized syringe is placed on a withdrawal pump (Harvard Instruments Model 600-900), and blood is withdrawn at a constant rate of 38.2 ml./min. Injection of indocyanine green dye[3] (CardioGreen®)** is made into the cardiac chambers or great vessels through the catheter positioned according to the information desired. Dye curves are recorded on photographic paper at a paper speed of 2.5 mm./sec. with time line markings at one second intervals using the Model ODC-8 Dye Curve Amplifier and the Electronics for Medicine PR 7 Recorder.

Calibration of the densitometer is performed after each cardiac catheterization by a modification of the method of Nicholson, Burchell, and Wood.[2] 30 ml. of arterial blood is withdrawn prior to the performance of dye curves into a heparinized flask. 10 ml. of this blood is used for a control of zero dye concentration. A stock solution is prepared by diluting 0.1 ml. of the dye (5 mgm./ml.) in 4.9 ml. of blood. One-half ml. of this stock solution is diluted in 4.5 ml. of blood to provide a concentration of 10 mgm. dye/L. After recording the zero dye in blood control and the 10 mgm./L. dilution, 2.5 ml. of the latter is diluted in 2.5 ml. of blood to record the 5 mgm./L. dilution. After each dilution is recorded, another dilution can be made in the same way until dilutions encompassing the dye curves recorded are made. By plotting centimeter deflection from zero concentration versus dilution in mgm. dye/L., a curve can be constructed from which other concentrations may be read after measuring indicator concentration in centimeters. It should be stressed that the sensitivity of the densitometer must remain unchanged for both the recording of the dye curves and the calibration thereafter.

Cardiac Output

An accurately measured amount of Cardio-Green dye (5 mgm. or 2.5 mgm.) is flushed through the catheter into the main pulmonary artery. Sampling is done through the system indi-

*Presented at staff meeting, Providence Memorial Hospital, El Paso, September, 1964.

**Hynson, Wescott, and Dunning, Inc., Baltimore, Maryland.

cated in Figure 1. To determine cardiac output, the indicator concentrations are measured at one second intervals to a point on the downslope equal to about 50 per cent of the peak concentration (C_P). The measurements in centimeters are converted to concentration in mgm./L. by reference to the calibration curve. The downslope concentrations in mgm./L. are then replotted on three cycle semilog paper and the line extended to a point equivalent to two per cent of the peak concentration. Values are read at one second intervals and all the concentrations read both from the curve and from the semi-log replot are added to give the sum of the concentrations (SC). If measurements are made at one second intervals, then the average concentration of the curve ($\overline{C}$) = SC/T where T is the time required for the recording of the first circulation of dye with recirculation excluded by semi-log replotting. The Stewart-Hamilton formula[4,5] for the determination of cardiac output then becomes 60 I/SC where I is the amount of dye in mgm. injected (Figure 2).

The process of semi-logarithmic replotting may be circumvented by a mathematical formula proposed and tested by Hoffman[6] for SC based on integration of the dye curve by parts with summation to infinity. Concentrations are measured at one second intervals to a point n at which the downslope begins its exponential decay. The concentration ½ second later (i.e. at n + ½) is measured (Ca). Another concentration is measured on the downslope (Cb). The logarithmic decrement of the curve (g) = $2.3 \frac{\log_{10} Ca - \log_{10} Cb}{Ta - Tb}$ in which the difference of the logarithms of any two concentrations on the downslope is divided by the difference in time at which they occur. The sum of concentrations (SC) = (sum to point n) + $(\frac{Cn + ½}{g})$. While electronic computors are available for cardiac output measurements, the Hoffman formula may provide a valid mathematical shortcut to the work of semi-log replotting where such a computer is not available.

Valvular Stenosis

Determination of cardiac output assumes great importance in the assessment of valvular stenosis. In 1951, Gorlin and Gorlin[8] proposed a hydraulic formula for the calculation of the area of a stenotic valve which indicates the basic inter-relationships of the valve area, pressure gradient across the valve, and flow rate across the valve. Measurement of the pressure gradient across the stenotic valve together with a measurement of cardiac output by the dye dilution technic at operation has been reported by Tannenbaum, Braunwald, and Morrow[9] to assess the significance of pressure gradients. The time required for recording cardiac output by dye-dilution technic (about one minute) is a definite advantage over the Fick method which requires considerably longer. The requirements for a steady state as well as practical considerations in limiting the time of ventricular puncture or presence of a catheter passed across a stenotic valve can more nearly be met by use of the dye-dilution technic.

Figure 3 illustrates the use of cardiac output determined by dye-dilution in conjunction with the aortic valve gradient to calculate the aortic valve area in a case of severe aortic stenosis. By combining these two measurements with the diagram from Gorlin[10] indicated in the figure, an understanding of the flow through, and the pressure gradients developed about a given valve may be obtained. When critical stenosis occurs, flow through the valve is limited, and increases in flow require increases in pressure gradient. Normal valve areas and critical stenosis are given below the for four cardiac valves. These then serve as a guide to the selection of patients for valvular surgery.

Valve	*Normal Area*	*Critical Stenotic Area*
Mitral	5 sq. cm.	1.2 sq. cm.[11]
Aortic	3-4 sq. cm.	0.5 — 0.8 sq. cm.[12]
Pulmonic	2-4 sq. cm.	0.8 sq. cm.[12]
Tricuspid	7 sq. cm.	1.5 sq. cm.[12]

Valvular Regurgitation

Assessment of the degree of valvular regurgitation continues to pose problems for the clinician. Progress in the development of valvular prostheses is requiring an assessment of the magnitude of valvular regurgitation in those patients who are symptomatic. While only the presence of disabling and progressive symptoms warrants the use of prosthetic replacement at this time,[14] dye-dilution technics may be of value to the surgeon in planning the type of operation and in selecting patients for surgery where a discrepancy between the symptoms and one of the means of evaluation (i.e. EKG, x-ray) arises.

In the presence of severe mitral regurgitation,

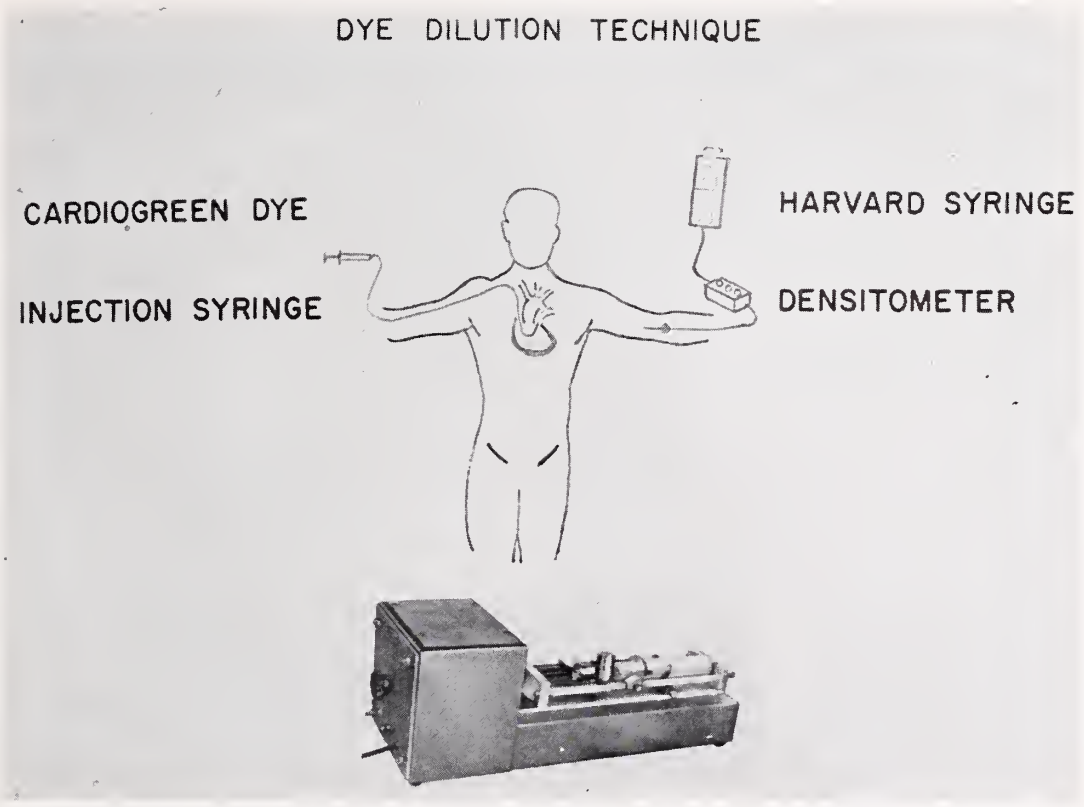


Figure 1. Diagram of assembly for dye-dilution studies during cardiac catheterization. In this and all subsequent figures, the injection and sampling facilities are shown on opposite sides of the figure for clarity. In practice, both injection and sampling are done through the same cutdown site. The densitometer leads to a recorder which is not shown in the figure.

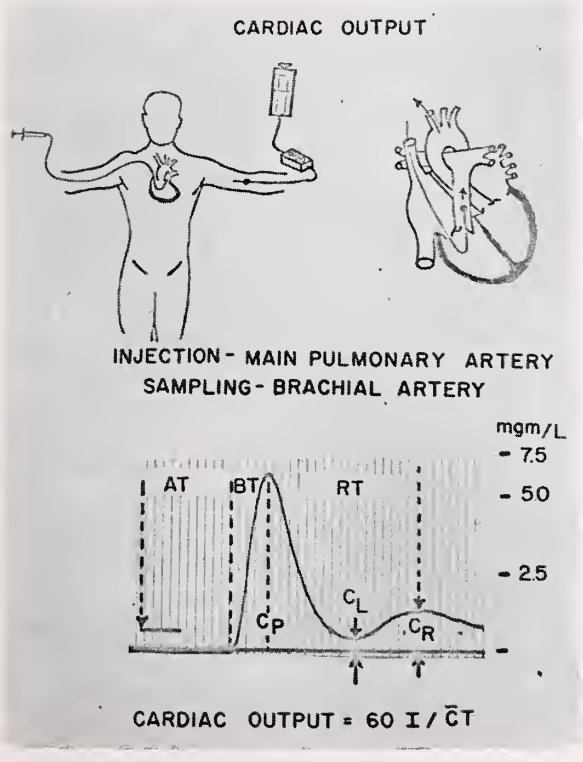


Figure 2. Cardiac output determination by dye-dilution technic, illustrating assembly, pathway of indicator through the heart, and a representative curve. AT—appearance time. BT—buildup time. RT—recirculation time. C_P—peak concentration. C_L—least concentration. C_R—maximum concentration of recirculation curve. I—milligrams of dye injected. $\bar{C}$—average concentration of dye. T—passage time of primary circulation curve after semilogarithmic extrapolation. Time lines are recorded at 1 second intervals. Calibration of densitometer is shown at the right of the curve in mgm./L.

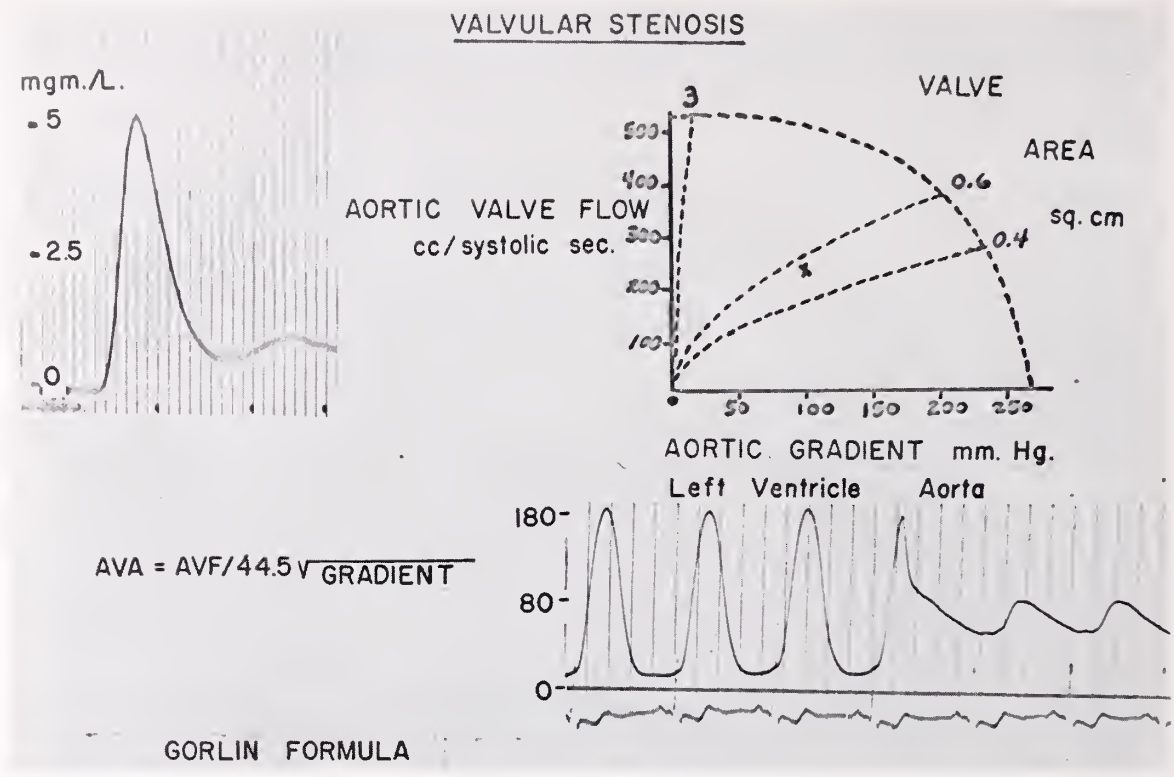


Figure 3. Assessment of the size of a stenotic aortic valve by the Gorlin formula. Illustrating dye-dilution curve for determining cardiac output, pressure change on withdrawal of catheter from left ventricle to aorta, and the relationship between pressure gradient, flow, and valve size. AVA—aortic valve area. AVF—aortic valve flow in systole.

the dye curve may be distorted by the reduction of the peak concentration, disproportionate prolongation of the disappearance slope, and absence of the systemic recirculation curve so that the C_L/C_R ratio is greater than one. The latter ratio when greater than 0.65 provides a useful method of separating cases with predominant or pure mitral regurgitation from those with predominant or pure mitral stenosis. Overlap of values into the range of predominant regurgitation (C_L/C_R greater than 0.65) may occur in patients categorized as having predominant mitral stenosis with significant regurgitation.[15] However, in practice, the latter would point to an open operation with the pump-oxygenator so that the correct surgical approach would be utilized.

Quantitation of the degree of regurgitation may be made by injecting dye downstream to the regurgitant valve with sampling both from the chamber receiving the regurgitant stream and a systemic artery[16, 17] or, alternatively, sampling from the chamber receiving the regurgitant stream after injections of equal amounts of dye into the region immediately downstream and the chamber or great vessel located upstream.[16] In the latter method, only a single densitometer is required, and if the densitometer attenuation remains unchanged between dye curves, both dye curves will have the same calibration. The ratio of the areas recorded after upstream and downstream injection represents the regurgitant fraction of dye. These areas may be measured with sufficient accuracy by the total triangle formula after extrapolation of the disappearance slope of both dye curves to a baseline of zero concentration on the photographic records. The formulas are indicated in Figure 4 with the divisor two being omitted from each area calculation since this cancels out in the ratio. The regurgitant fraction of dye correlates closely with the ratio of regurgitant volume to total stroke volume or of regurgitant flow to total ventricular output, the latter ratios indicating the regurgitant fraction of blood.[17] Thus a regurgitant fraction of 50 per cent indicates that the regurgitant flow is equal to the systemic flow available for tissue use,

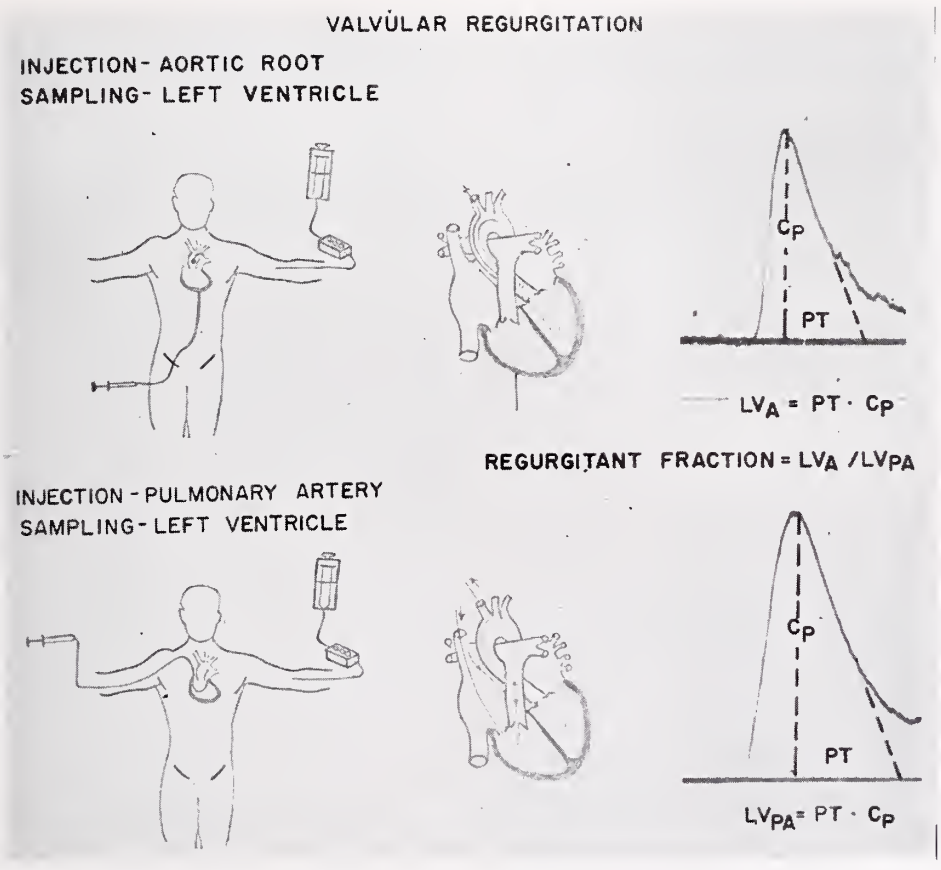


Figure 4. Determination of the regurgitant fraction in aortic regurgitation. Regurgitant fraction equals 55%, indicating that regurgitation is greater than systemic flow. LV_A—area of curve obtained by left ventricular sampling after aortic root injection. LV_{PA}—area of curve obtained by left ventricular sampling after pulmonary artery sampling. PT—passage time. C_P—peak concentration. The divisor 2 is omitted from the area determinations because it cancels in the final ratio.

doubling the volume load on the ventricle. An extensive clinical[18, 19, 20] as well as experimental[16, 17] experience has confirmed the utility of this upstream sampling technic in evaluating the degree of valvular regurgitation. Arbitrarily, the degree of valvular regurgitation is graded as slight with a regurgitant fraction of less than 20 per cent, moderate with 20-40 per cent regurgitation, and severe with regurgitation of 40 per cent or more by this method. At the present time, surgery for the correction of valvular regurgitation at the aortic or mitral valves is recommended for patients with progressive and disabling symptoms (Class III and IV) with severe valvular regurgitation where no contraindication exists.[21] Patients with severe aortic regurgitation manifesting the nocturnal anginal syndrome as discussed by Bland and Wheeler,[22] and Griep[23] are considered for surgery because of the poor prognosis of this syndrome despite the fact that criteria for inclusion in Class III and IV are not met.

Right to Left Shunt

Swan, Zapata-Diaz, and Wood[24] reported the dye-dilution method used for estimating the magnitude of right to left shunts. Dye is injected at or upstream to the site of the right to left shunt and is sampled from a systemic artery. In the presence of a cardiac defect which permits right to left shunting of blood, dye will bypass the lungs and appear early at the sampling site. Dye traversing the pulmonary circulation will appear at the time normally expected so that a distorted upstroke or double-humped dye curve with an early appearance time will be recorded. In assessing the appearance time of a dye curve, the normals for a

radial sampling site reported by Carter, Swan, and Wood[25] are a useful guide.

Since there is a time delay due to the dead space of the densitometer sampling system, a correction must be made by dividing the dead space volume of the densitometer sampling system (V_{DSS}) measured from needle tip to detecting element in the densitometer by the flow rate of blood withdrawn through the cuvet system (Q_{DSS}).[25] An alternative formula for correction of the time delay due to the dead space in the densitometer sampling is 0.6 (V_{DSS}/Q_{DSS}) where 0.6 is an empirically determined factor to correct for the fact that laminar flow occurs in at least portions of the system.[16] The appearance time is corrected for this time delay by moving the instant of injection toward the dye curve by the duration of the time delay (in Figure 5, this amounted to three sec.). The dye curve is then regarded as two triangles, the first representing the right to left shunt, the second representing blood traversing the pulmonary circulation, and the sum of the two representing total systemic flow. The areas of the two portions of the curves are estimated by the forward triangle formula. The buildup time of the second portion of the curve is estimated at 0.46 of the MCT (maximal concentration time measured from the second peak to the arrow indicated on Figure 5).[1] The right to left shunt as a per cent of total systemic flow is given by the formula illustrated in Figure 5. Since shunts comprising 25 per cent of the systemic flow may occur with arterial oxygen saturation around 90 per cent,[24] and since definite cyanosis is not usually recognized until the arterial oxygen saturation falls to about 75 per cent,[26] this dye dilution method with superior vena caval injection at catheterization insures that a right to left shunt across the atrial or ventricular septum will not be overlooked. Furthermore, in the assessment of patients with atrial septal defects for surgical closure, McGoon and associates have indicated that the magnitude of the right to left shunt is one factor in assessing the risk. A right to left shunt 10 per cent or more of systemic flow was associated with a 50 per cent mortality in the closure of atrial spetal defects in their series.[27] This dye-dilution technic then proves the existence of a defect between the right and left heart, localizes the site of the defect, and provides a method of estimating the magnitude of the shunt, independent of calculations using oxygen data.

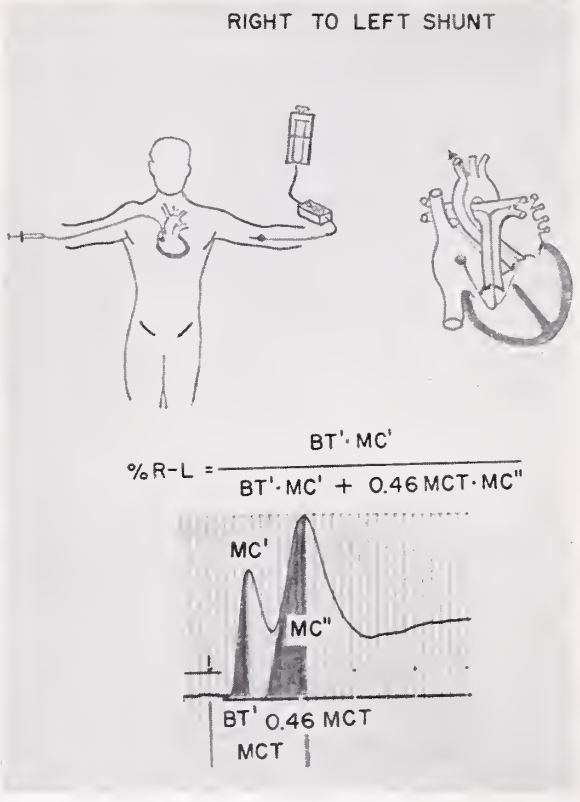


Figure 5. Assessment of a right to left shunt through a ventricular septal defect by the method of Swan, Zapata-Diaz, and Wood. Arrow indicates the instant of injection corrected for time delay due to the dead space of the sampling system. BT'—buildup time of shunt curve. MC'—maximum concentration of shunt curve. MC"—maximum concentration of dye traversing the pulmonary circulation. MCT—interval between injection and MC". The use of the constant 0.46 is explained in the original article by Swan.[24] %R-L—right to left shunt as per cent of systemic flow.

Left to Right Shunt

Problems often arise in assessing the magnitude of a left to right shunt in patients with cardiac septal defects. Carter, Bajec, Yannicelli, and Wood[28] have reported the method of detecting the presence of a left to right shunt and estimating its magnitude from an arterial dilution curve recorded after injection into the main pulmonary artery. In this method, the concentrations are measured at the peak of concentration, and again at one and two buildup times from the peak. The correlation of the disappearance ratios ($C_P/CP+$ BT and C_P+_{2BT}) with the magnitude of the left to right shunts by the Fick method and by simultaneous venous and systemic dilution curves forms the basis of the regression equations formulated by Carter and reproduced in Figure 6. Evaluation of

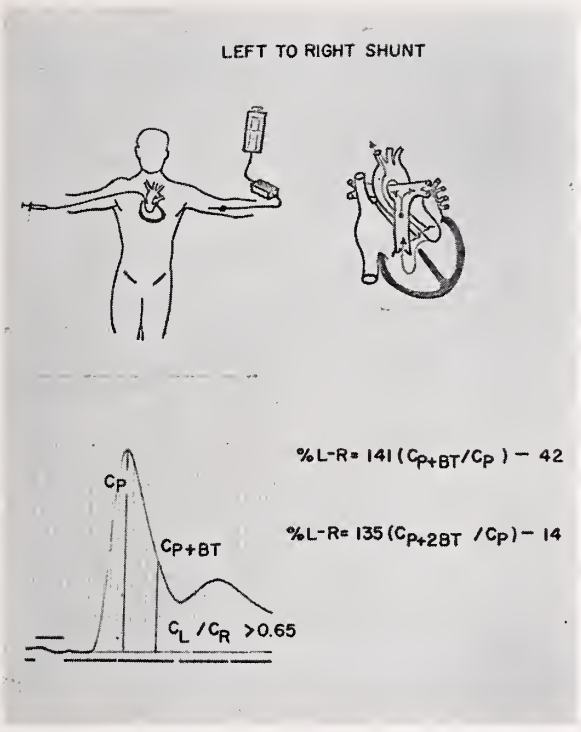


Figure 6. Assessment of left to right shunt through a ventricular septal defect by the regression equation of Carter et al.[28] CP—peak concentration. $C_P + BT$—concentration at one buildup time beyound the peak. $C_P + 2BT$—concentration at two buildup times beyond the peak. C_L—least concentration. C_R—maximum concentration of systemic recirculation curve. %L-R—left to right shunt as a per cent of pulmonary flow.

the C_L/C_R ratios provides a means of detecting small distortions of the disappearance slope. Normally, the C_L/C_R ratio ranges from 0.26-0.60. Patients with left to right shunts under 35 per cent with one exception showed C_L/C_R ratios of 0.65 or greater. With left to right shunts equal to or greater than 35 per cent of the pulmonary flow, the C_L/C_R ratios were 0.90 to greater than one.[28]

This dye-dilution technic provides an independent method for the detection of a left to right shunt where data obtained from analyses of right heart blood for oxygen saturation and content are equivocal. Since the Carter formulas indicate that per cent of pulmonary flow composed of shunted blood, a 50 per cent shunt would double the volume load on both ventricles in the presence of a ventricular septal defect, and would double the volume load on the right ventricle in the presence of an atrial septal defect. In selecting patients for surgery, a shunt of this magnitude would be one indication for closure of the septal defect.[29]

Summary

The dye-dilution technic is a useful adjunct to cardiac catheterization and to the practice of clinical cardiology, especially when the selection of patients for surgical treatment is undertaken. The magnitude of the hemodynamic defect must be assessed, and dye-dilution technics play an important part in this assessment. Some of the technics available are reviewed, together with the formulas used in estimating the hemodynamic abnormality.

Acknowledgment

The technical assistance of Mrs. Joan Ballard, R.N., Mrs. Nancy Bryant, R.N., Mrs. Shirley Engel, R.N., and Mrs. Katherine Watts, R.N., in the performance of these studies is gratefully acknowledged. The cooperation of members of the Radiology Department and other hospital personnel is also appreciated.

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Medical Assistants Meet In El Paso

A one-day Traveling Educational Symposium for Medical Assistants, sponsored by the Texas Medical Assistants Association through a grant of the Texas Medical Association, was held in El Paso March 7, 1965.

Physicians participating were Dr. Laurance N. Nickey, Dr. H. D. Garrett, and Dr. Russell L. Deter, all of El Paso. Others were Miss Jo Estrada, San Antonio, President-Elect of the Texas Medical Assistants Association, Jerry Harris, El Paso, Managing Director of Creditors Service Bureau, Medical Arts Division, C. Dean Davis, legal counsel for the TMA, Bob Reid, El Paso, Mott—Reid—McFall Public Relations and Advertising, Mrs. Sue Coulter, President of El Paso County Medical Assistants Society, and Mrs. Phyllis Porth, El Paso, member of the Traveling Educational Symposium Committee. A Wyeth Laboratories film on sterilization procedures for the medical office was shown by H. A. Kenady, El Paso, Wyeth territory manager.

Subjects covered in the Symposium were public relations, billing and collecting, legal problems, medical ethics, professional growth, and the doctor, medical assistant and patient relationship. New members of the El Paso County Medical Assistants advisory committee are Dr. Deter, chairman, Dr. Garrett, Dr. Wm. Arnold Pitchford, and Dr. Glen E. Furr.

Medical Records

I

I don't think that I shall ever see
A Chart as lovely as a bumble bee.
A Chart whose thorny barbs are spread
To prick the skin of a weary head.
A Chart whose many pages are stacked
In confusing order to the brain so racked.
A Chart whose hungry Title Page looks up—
The tentacles of an octopus, a huge suction cup.

II

With Progress Reports and a Discharge Note—
A History and Physical to get your goat.
With Lab Reports and X-Ray film,
Repeatedly read as the light grows dim.
With Nurses Notes and Anesthesia Records
Giving visual spots like a brace of leopards.
With Surgery Records and Path Reports
Producing evidence dangerous—and few retorts.
With M.A.P. and P.A.S.; full critics with domain
To judge your ability to continue and remain.
With Medical Records to hound and prod—
'Til the desire is strong to be under the sod.

III

And the patient—poor soul—his mind sublime,
Blissfully believed we gave him our time.
The time to visit, to study and examine;
To palpate and probe for the cause undermine.
The time to touch with fingers that hear
The sound of death to a life so dear.
The time to prescribe the drug with might,
To bring healthy life closer to sight.

IV

Alas! The time thus urgently needed
Escaped us all like the tide receded.
With gasping trust the patient dies—.
Soon, the marker reads, "Beneath here lies—."
But—the Chart moves on in endless ways;
The Autopsy Report takes days and days.
In discussion groups the Staff does ponder
The value of life both here and yonder.
With Records listening, and the Board on hand,
The poor patient's Chart is cleared of bad sand.

V

Thus, the Chart is passed with a word so brief,
While the poor patient's wife is stricken with grief.
The patient is lost, but the Records are clear;
We regret the life but Statistics remain dear.
Quick! A new patient is due! Assemble the Chart!
We must begin his program right from the start!
For—the sick will die; deadly wounds are not new;
But—look at their Charts!—so nice to review!

—W. L. Minton, M.D., Lovington, New Mexico

How Much Will Health Care Cost or How High The Moon?*

Omar Legant, M.D., *Albuquerque*
President, New Mexico Medical Society

The philosophy of the electorate as expressed November 3rd may be summarized in one word—MORE. The Americans for Democratic Action is now on record for a program of "full hospital and medical care for the elderly and ultimately the whole population".

Disregarded is the cost entailed in attempting to satisfy such limitless desires. But it's always open season for sniping at the price tag.

Swimming against the relentless tide are a few groups, like organized medicine, who honestly believe we should continue to concern ourselves with ways and means of lowering the cost of health care. Your peripatetic president has recently attended three AMA sponsored conferences with representation from all groups with a stake in the problem—including the consumer. The themes were conservation of the health care dollar; hospital costs and patient expenditures; area-wide hospital facilities planning.

Since the recommendations which emerged, if implemented, will greatly influence the way physicians practice, we shall report the proceedings in some detail.

More Responsibility

All recommendations made require physician participation and cooperation. The various proposals saddle with additional committee assignments the same 15 to 20 percent of the practicing medical profession whose hypertrophied sense of responsibility leads them to accept membership on the tissue committees, infection committees, record committees and other appurtenances of hospital life in addition to being active in their medical societies, on Blue Shield Boards and the like. These duties of course are over and above their primary purpose—taking care of sick folks—which entails a 65-hour work week. This minority of the profession clearly understands that if we physicians dodge such responsibilities others, not quite so burdened, will assume them for us.

Let us begin on a chilling note—the attitude of Big Unions. Their representatives are not horny handed toilers as you might expect but Ph.D.'s with a social welfare background who are not above using a little demagoguery along with Sweeping Assertions and Unwarranted Assumptions. They refer to current insurance programs disparagingly as "sickness insurance" which become operative when the subscriber is sick enough to be hospitalized. What they desire is "health insurance" which they believe should cover everything including the visit to the doctor, the routine physical checkup, the immunization shots, etc. These items, some of us feel, are entirely within people's ability to budget for themselves. Labor wants first dollar coverage, without such tedious deterrents to usage as deductibles and co-insurance. The costs of such coverage are simply something to be unloaded on the employer at the next session around the bargaining table. The fact that the employer will pass it on to the public giving inflation another boost was not mentioned. These representatives want quality control of medical practice—board certification being the sine qua non of quality medicine. Fee-for-service and solo practice are going out, they say, and should be replaced by more full time salaried hospital-employed physi-

*Reprinted from The New Mexico Medical Society NEWSletter, January, 1965.

cians. Considering the political power they represent this could be the Wave of the Future for medical practice.

Recommendations

Several recommendations were made by the representatives of hospital administration, insurance carriers, and the medical profession to reduce the cost of hospital care. 1. Cost analysis and publication of expenditures by individual hospitals. 2. Early transfer from general hospital to less expensive convalescent home or progressive self-care unit. 3. Hospital utilization committees to establish norms for duration of hospital stays for various ailments; to investigate possible instances of over-usage. 4. Restriction of prescription to the hospital formulary and by generic name preferably. In one 500-bed hospital a saving of $180,000 on the pharmaceutical bill in one year was accomplished by this means. 5. Area-wide pre-construction hospital facilities planning on a voluntary basis. 6. Increased out-of-hospital benefits by health insurance carriers. Why should the ambulatory patient with a diagnostic problem run up a two to three-day hospital bill while he's being prepared for various tests, being examined, the results reported and finally being read by his physician? This could be done as well prior to hospitalization. The results of the test might even indicate no treatment necessitating hospitalization is necessary.

However, even if all these suggestions were implemented, we expect hospital costs will continue to rise inexorably. Health care expenditures have risen 287 per cent since 1946. The prime culprit is rising hospital costs which now takes the major share of the health care dollar. By far the greatest single factor in rising hospital per diem costs is wages—an increase of 333 per cent, accounting for 65 per cent of hospital expenditures. Despite this there is still about a 31 per cent gap between hospital wages and the average wage in industry. We physicians believe that hospital workers' efforts to upgrade a long standing low pay scale should be supported not discouraged. We should not expect to continue employing people in hospitals at a low wage by an appeal to their humanitarian instincts.

Speaking personally, I feel all concerned in the health industry are obligated to practice efficiently, to provide high quality care and to inform the public as to the reasons for the spiraling costs of such care. I believe it is then the public's obligation to finance the all-encompassing benefits it has indicated it desires.

I believe it is the duty of every doctor, be he an internist, a radiologist, even a dermatologist, in fact whatever he may be, if he is interested in human welfare that he pay attention to the fact that no American couple should have more than two and a half babies, and if he doesn't know how to make it possible for them to limit their output to that then he ought to send them to somebody who does. — *Dr. John Rock, quoted in New York Medicine.*

We can't think who that would be. — *quoted from The New Yorker.*

Coming Meetings

Arizona Chest Disease Symposium, Ramada Inn, Tucson, April 4, 1965.

17th Annual Scientific Assembly of the American Academy of General Practice, Civic Auditorium, San Francisco, April 12-16, 1965.

Third National ISA (Instrument Society of America) Biochemical Sciences Instrumentation Symposium, Statler Hotel, Dallas, April 19-21, 1965.

23rd Annual Meeting, U.S.-Mexico Border Public Health Ass'n., Los Angeles, April 26-29, 1965.

Annual Session, Texas Medical Association, San Antonio, April 29-May 2, 1965. C. Lincoln Williston, 1801 N. Lamar, Austin, Exec. Secy.

83rd Annual Meeting of the New Mexico Medical Society and 12th Biennial Meeting of the Rocky Mountain Medical Conference, La Fonda, Santa Fe, May 9-15, 1965.

Eighth Annual Ruidoso Summer Clinic, sponsored by the New Mexico Chapter of American Academy of General Practice, Ruidoso, N. M., July 19-22, 1965. Headquarters: Chaparral Motel.

62nd Annual Meeting of the Western Association of Railway Surgeons, Alameda Hotel, Avenida Juarez 50, Mexico City, October 27-30, 1965.

15th Annual Meeting of the Southwest Obstetrical and Gynecological Society, Arizona Inn, Tucson, Oct. 28-30, 1965.

47th Annual Meeting, Southwestern Medical Association, Sheraton Motor Inn, El Paso, November 4-6, 1965.



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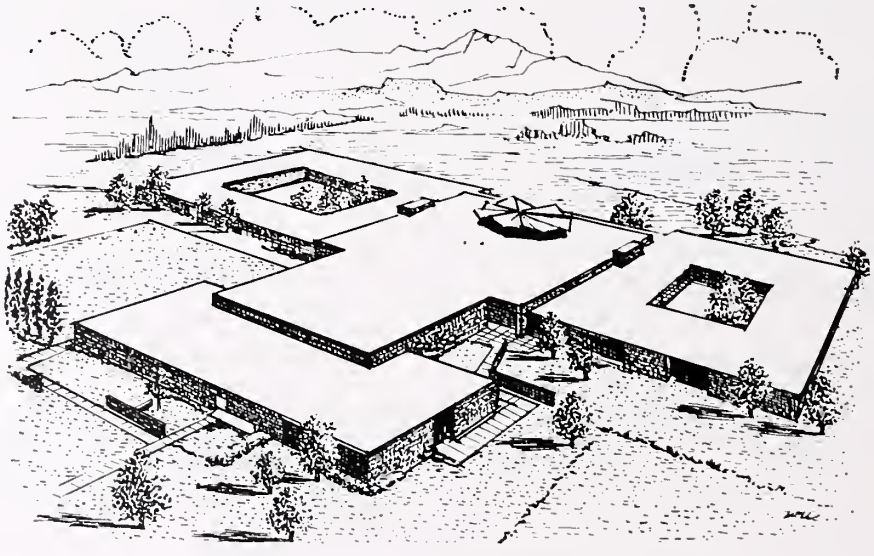
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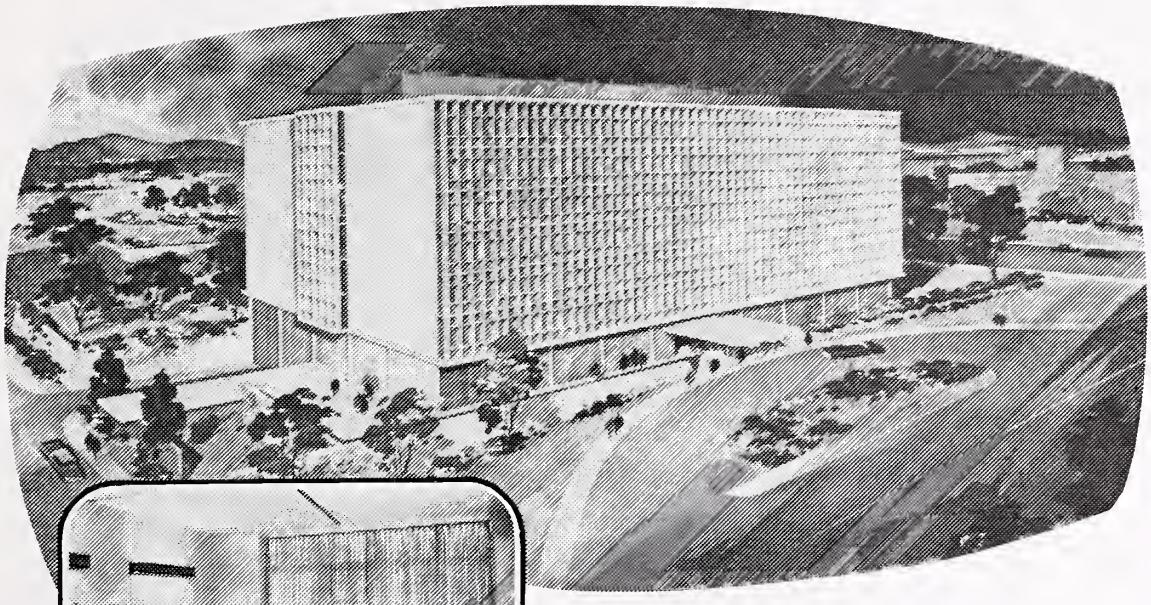


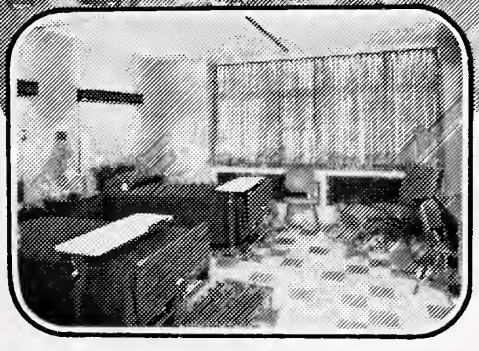
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Insomnia often presents a treatment problem. Although sedatives may give limited relief, they frequently tend to deepen depression. Antidepressants, particularly stimulants and "energizers", may often aggravate insomnia because of their excitatory effect or their inability to alleviate the anxiety which frequently accompanies depression. ▪ With 'Deprol', early relief of associated insomnia can be a rewarding first step in treatment of depression. 'Deprol' usually restores normal sleep by relieving the associated anxiety and tension which often cause or intensify insomnia while it acts to reduce the underlying depression.

Indications: 'Deprol' is useful in the management of depression, both acute (reactive) and chronic. It is particularly useful in the less severe depressions and where the depression is accompanied by anxiety, insomnia, agitation, or rumination. It is also useful for management of depression and associated anxiety accompanying or related to organic illnesses. **Contraindications:** Benactyzine hydrochloride is contraindicated in glaucoma. Previous allergic or idiosyncratic reactions to meprobamate contraindicate subsequent use. **Precautions:** *Meprobamate:* Careful supervision of dose and amounts prescribed is advised. Consider possibility of dependence, particularly in patients with history of drug or alcohol addiction; withdraw gradually after use for weeks or months at excessive dosage. Abrupt withdrawal may precipitate recurrence of pre-existing symptoms, or withdrawal reactions including, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, the dose should be reduced and operation of motor vehicles or machinery or other activity requiring alertness should be avoided if these symptoms are present. Effects of excessive alcohol may possibly be increased by meprobamate. Grand mal seizures may be precipitated in persons suffering from both grand and petit mal. Prescribe cautiously and in small quantities to patients with suicidal tendencies. **Side effects:** Side effects associated with recommended doses of 'Deprol' have been infrequent and usually easily controlled. These have included drowsiness and occasional dizziness, headache, infrequent skin rash, dryness of mouth, gastrointestinal symptoms, paresthesias, rare instances of syncope, and one case each of severe nervousness, loss of power of concentration, and withdrawal reaction (status epilepticus) after sudden discontinuation of excessive dosage. *Benactyzine hydrochloride:* Benactyzine hydrochloride, particularly in high dosage, may produce dizziness, thought-blocking, a sense of depersonalization, aggravation of anxiety or disturbance of sleep patterns, and a subjective feeling of muscle relaxation, as well as anticholinergic effects such as blurred vision, dryness of mouth, or failure of visual accommodation. Other reported side effects have included gastric distress, allergic response, ataxia, and euphoria. *Meprobamate:* Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse. **Dosage:** Usual starting dose, one tablet three or four times daily. May be increased gradually to six tablets daily and gradually reduced to maintenance levels upon establishment of relief. Doses above six tablets daily are not recommended even though higher doses have been used by some clinicians to control depression and in chronic psychotic patients. **Supplied:** Light-pink, scored tablets, each containing meprobamate 400 mg. and benactyzine hydrochloride 1 mg. *Before prescribing, consult package circular.*

CD-4092

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meprobamate 400 mg.
+ benactyzine hydrochloride 1 mg.

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Southwestern MEDICINE

Official Journal of the Southwestern Medical Association,
The Western Association of Railway Surgeons, Southwestern Dermatological Society,
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and El Paso County Medical Society

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OCT 18 1965

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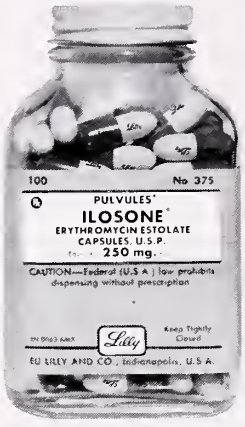
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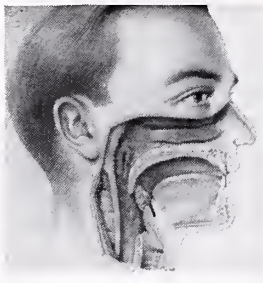
VOL. 46, NO. 4

April, 1965

Founded 1916

An added margin





to meet an extra challenge

Test-tube activity of a drug is only one indication of its effectiveness. More important is the amount of antibacterial activity at the site of infection.

Ilosone® produces peak levels of antibacterial activity *two* to *four* times those of other erythromycin preparations. Furthermore, it attains them earlier and maintains them longer.

Contraindications: Ilosone is contraindicated in patients with a known history of sensitivity to this drug and in those with preexisting liver disease or dysfunction.

Side-Effects: Even though Ilosone is the most active oral form of erythromycin, the incidence of side-effects is low. Infrequent cases of drug idiosyncrasy, manifested by a form of intrahepatic cholestatic jaundice, have been reported. There have been no known fatal or definite residual effects. Gastro-intestinal disturbances not associated with hepatic effects are observed in a small proportion of patients as a result of a local stimulating action of Ilosone on the alimentary tract. Although allergic manifestations are uncommon with the use of erythromycin, there have been occasional reports of urticaria, skin eruptions, and, on rare occasions, anaphylaxis.

Dosage: *Children under 25 pounds*—5 mg. per pound of body weight every six hours. *Children 25 to 50 pounds*—125 mg. every six hours. *Adults and children over 50 pounds*—250 mg. every six hours. For severe infections, these dosages may be doubled.

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Recommended Dosage of Lomotil Liquid to Control Diarrhea

File: Diarrhea

LOMOTIL®

Age	INITIAL LOMOTIL LIQUID DOSAGE*
3-6 mo.	½ tsp. t.i.d. (3 mg.)
6-12 mo.	½ tsp. q.i.d. (4 mg.)
1-2 yr.	½ tsp. 5 times daily (5 mg.)
2-5 yr.	1 tsp. t.i.d. (6 mg.)
5-8 yr.	1 tsp. q.i.d. (8 mg.)
8-12 yr.	1 tsp. 5 times daily (10 mg.)
Adult	2 tsp. 5 times daily (20 mg.) (or 2 tablets q.i.d.)

*Based on 4 cc. per average teaspoonful.
Note: After diarrhea is controlled the initial dosage can usually be reduced to meet the requirements of the individual patient.

LOMOTIL® TABLETS/LIQUID

Each tablet and each 5 cc. of liquid contains:
diphenoxylate hydrochloride .....2.5 mg.
(Warning: May be habit forming)
atropine sulfate .....0.025 mg.

Precautions
Lomotil is an exempt narcotic preparation of very low addictive potential. Recommended dosages should not be exceeded. Lomotil should be used with caution in patients with impaired liver function and in patients taking addicting drugs or barbiturates.

Cautions and Side Effects
Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness and insomnia.

Lomotil is a brand of diphenoxylate hydrochloride with atropine sulfate; the subtherapeutic amount of atropine is added to discourage deliberate overdosage.

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Southwestern Medicine

The U. S.-Mexico Regional Medical Journal Serving West Texas, New Mexico, Arizona, Nevada and Northern Mexico

Official Journal of

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VOL. 46 APRIL, 1965 NO. 4

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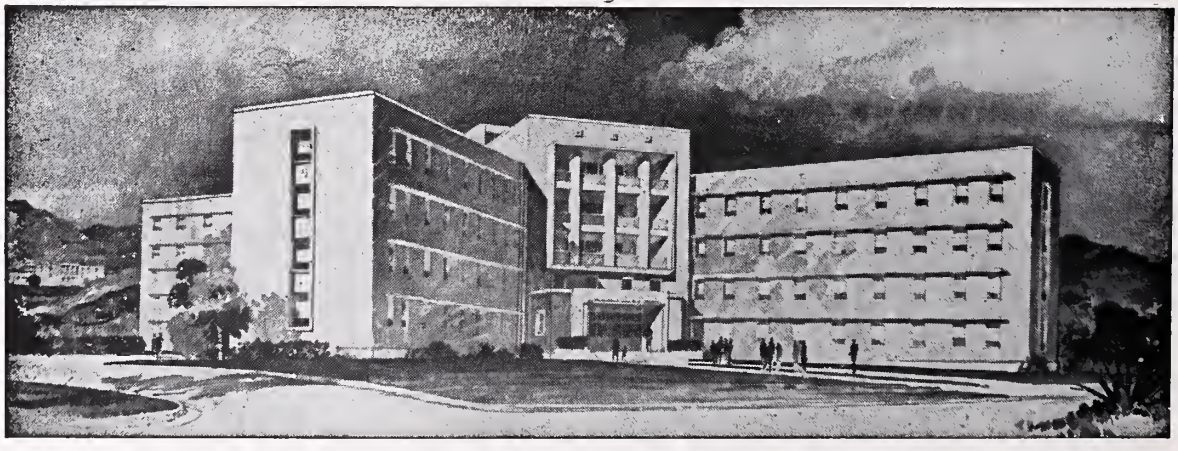
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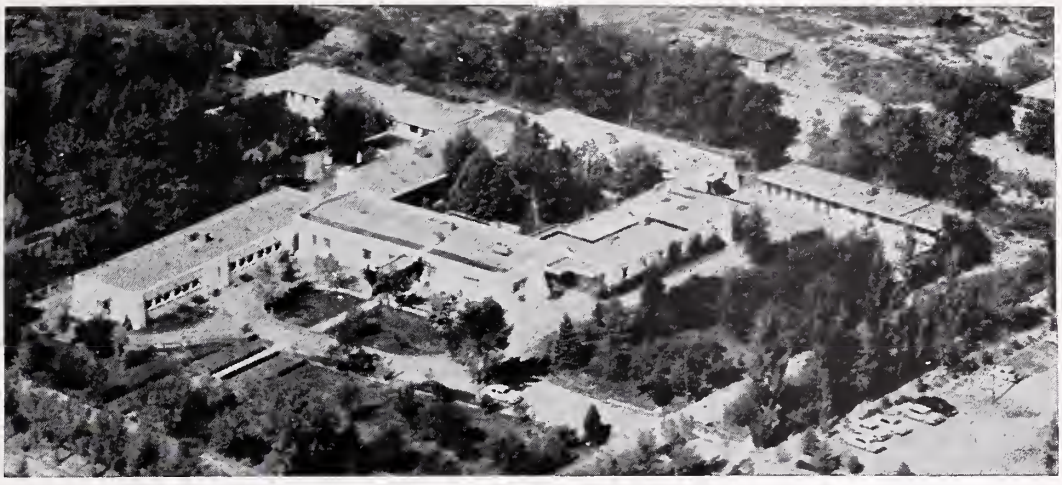
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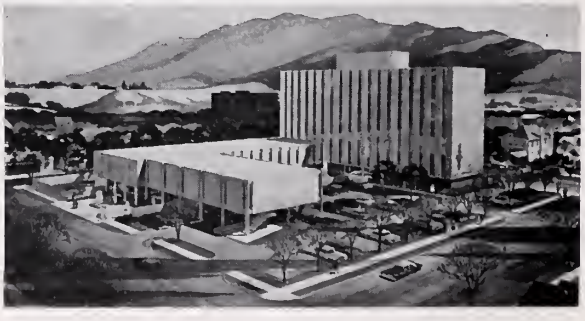
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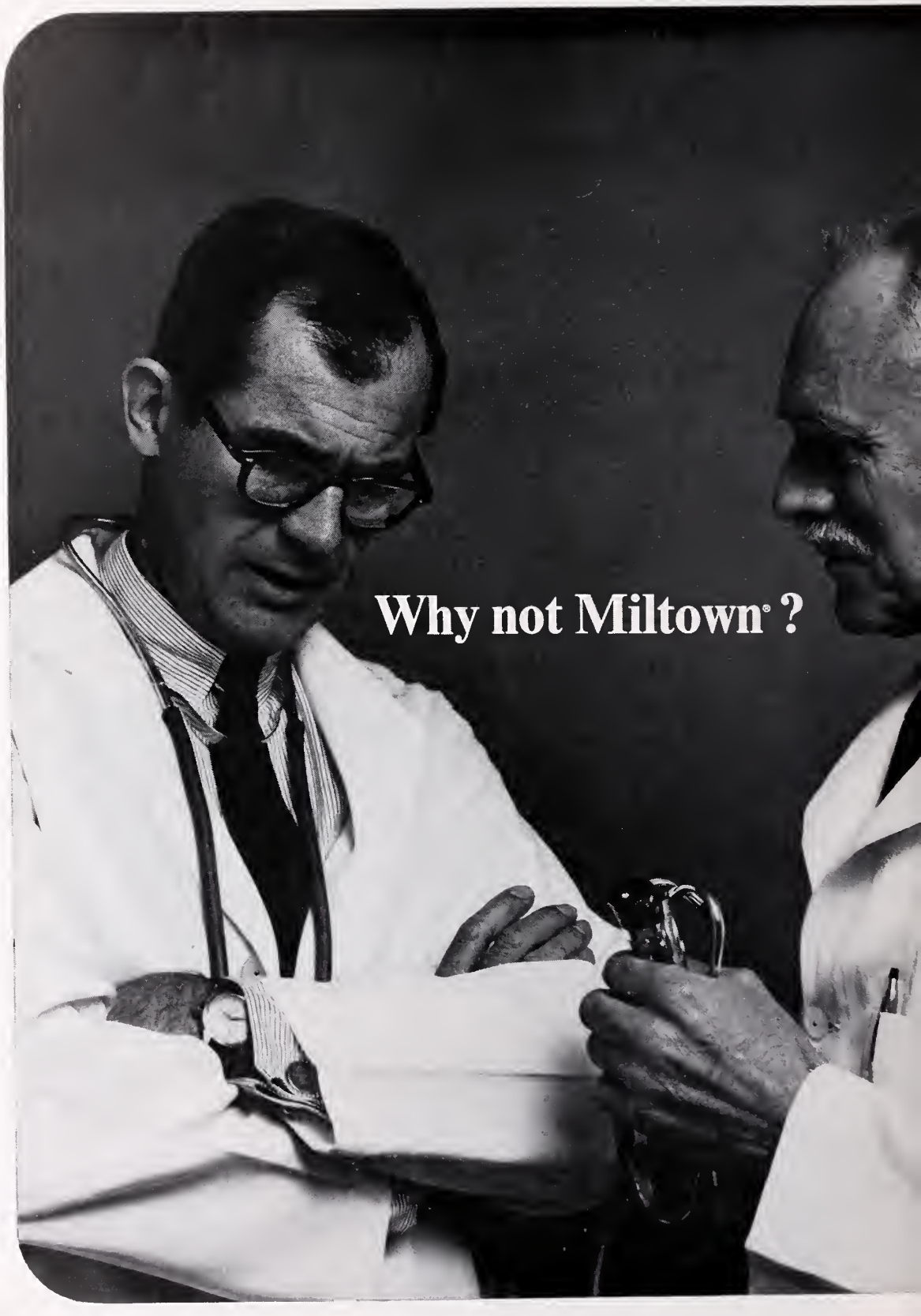
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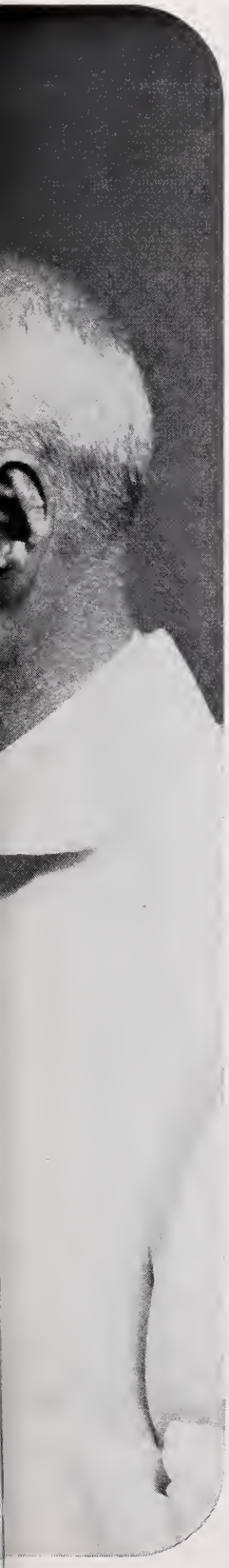
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- **Clinicians throughout the world consider meprobamate a therapeutic standard in the management of anxiety and tension.**
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Indications: 'Miltown' (meprobamate) is effective in relief of anxiety and tension states. Also as adjunctive therapy when anxiety may be a causative or otherwise disturbing factor. Although not a hypnotic, 'Miltown' fosters normal sleep through both its anti-anxiety and muscle-relaxant properties.

Contraindications: Previous allergic or idiosyncratic reactions to meprobamate or meprobamate-containing drugs.

Precautions: Careful supervision of dose and amounts prescribed is advised. Consider possibility of dependence, particularly in patients with history of drug or alcohol addiction; withdraw gradually after use for weeks or months at excessive dosage. Abrupt withdrawal may precipitate recurrence of pre-existing symptoms, or withdrawal reactions including, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, the dose should be reduced and operation of motor vehicles or machinery or other activity requiring alertness should be avoided if these symptoms are present. Effects of excessive alcohol may possibly be increased by meprobamate. Grand mal seizures may be precipitated in persons suffering from both grand and petit mal. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side effects: Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

Usual adult dosage: One or two 400 mg. tablets three times daily. Doses above 2400 mg. daily are not recommended.

Supplied: In two strengths: 400 mg. scored tablets and 200 mg. coated tablets.

Before prescribing, consult package circular.

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(meprobamate)

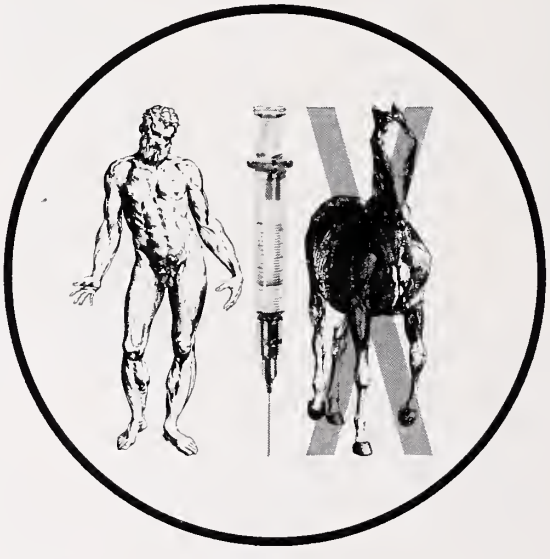
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Hyper-Tet is a gamma globulin fraction of venous blood from hyperimmunized humans and contains no heterologous protein. Far fewer units of Hyper-Tet are required for prophylaxis than with equine or bovine antitoxin. Recent studies[1-3] show these lower dosages of tetanus immune globulin (human) established protective levels well above the recommended immunity level[4] for up to 5 weeks.

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The tetanus antitoxin without horse serum and its reactions

Hyper-Tet™ [TETANUS IMMUNE GLOBULIN—HUMAN]

Hyper-Tet is available in 250 and 500 unit vials. A 250 unit dose is now regularly used in routine prophylactic cases. In cases where the injury is severe and where the risk of potential tetanus infection is higher, a dose in excess of 250 units may be indicated and antibiotic prophylaxis may also be advisable.[5]

Side Effects and Precautions: The likelihood of anaphylactoid or serum reactions due to intramuscular injection of gamma globulin is remote. Very rare serious reactions have been reported, however, and their extreme rarity makes it impossible to predict their occurrence. Slight soreness at and over the injection site may be noted. Do not give intravenously. There are no known contraindications.

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References: 1. Rubbo, S. D., and Suri, J. C.: Brit. M.J. *2*:79 (July 14) 1963. 2. Rubinstein, H. M.: Am. J. Hyg. *76*:276, 1962. 3. McComb, J. A.: New England J. Med. *270*:175 (Jan. 23) 1964. 4. Effective tetanus protective level established by Sir David Bruce. 5. Editorial: Tetanus Immunization, JAMA *161*:883, 1956.

Announce Program For N. M. Medical Society— Rocky Mountain Meeting

The complete program for the 83rd Annual Meeting of the New Mexico Medical Society and the 12th Biennial Meeting of the Rocky Mountain Medical Conference in Santa Fe, May 9-15, 1965, has been announced by Dr. Omar Legant, Albuquerque, President of the New Mexico Society.

Speakers will be:

Oscar Auerbach, M.D., Senior Medical Investigator, Veterans Administration Hospital, East Orange, New Jersey; Professor of Pathology, New York Medical College, New York.

Alexander Simon, M.D., Superintendent and Medical Director, The Langley Porter Neuropsychiatric Institute; Professor and Chairman of the Department of Psychiatry, University of California School of Medicine, San Francisco.

Lester R. Dragstedt, M.D., Research Professor of Surgery, University of Florida; Recipient of the Distinguished Service Medal of the American Medical Association.

Norman E. Shumway, M.D., Associate Professor of Surgery and Chief, Division of Cardiovascular Surgery, Stanford University.

Bernard I. Lewis, M.D., Internist, Metabolic Section, Palo Alto Medical Clinic; Assistant Clinical Professor of Medicine, Stanford University.

Edward Stainbrook, M.D., Professor and Chairman, Department of Psychiatry, University of Southern California School of Medicine; Chief Psychiatrist, Los Angeles County General Hospital.

Keith Reemtsma, M.D., Associate Professor of Surgery, Tulane University School of Medicine.

Audrey K. Brown, M.D., Associate Professor of Pediatrics, University of Virginia; Director, Pediatric Hematology Clinic, University of Virginia Hospital.

Alfred J. Bollet, M.D., Associate Professor of Internal Medicine and Preventive Medicine; Director, Rheumatic Disease Research and Training Program and Chronic Disease Unit, University of Virginia.

Hollis G. Boren, M.D., Director, Trudeau Foundation, Saranac Lake, New York.

William D. Cochran, M.D., Director, Maternal-Infant Health Program, Boston.

Brown M. Dobyns, M.D., Professor of Surgery, Western Reserve University.

Carl B. Pollock, M.D., Psychiatry Department, University of Colorado School of Medicine.

Members of the Rocky Mountain Medical Conference are from Colorado, Montana, Nevada, New Mexico, Utah, and Wyoming.

Headquarters for the meeting will be at the La Fonda. Registration is open to any Doctor of Medicine. Registration is not limited to physicians within the six states which participate in the Conference. The registration fee is $10. No fee will be charged physician members of the Armed Forces in uniform and nurses.

Officers of the New Mexico Society are, in addition to Dr. Legant, Dr. Robert P. Beaudette, Raton, President-Elect; Dr. Thomas Lyle Carr, Albuquerque, Vice-President; and Dr. Hugh B. Woodward, Albuquerque, Secretary-Treasurer.

The complete program follows:

PROGRAM

Sunday, May 9

2:00 p.m. Council Meeting
Coronado Room, La Fonda

7:00 p.m. Council Dinner

Monday, May 10

1:00 p.m. Delegates' Registration

2:00 p.m. Delegates' Meeting
Santa Fe Room, La Fonda

3:30 p.m. Reference Committee Meetings
Santa Fe Room
New Mexican Room
Coronado Room

Tuesday, May 11

9:00 a.m.-12:00 noon Orientation Course
Santa Fe Room

12:30 p.m. Luncheon for New Members
Coronado Room

2:30 p.m. Delegates' Meeting

Wednesday A.M., May 12

Presiding: Omar Legant, M.D., Albuquerque, President
New Mexico Medical Society

8:45-9:00 a.m. Invocation and Welcome

9:00-9:30 a.m. "The Battered Child Syndrome"
Carl B. Pollock, M.D., Denver

9:30-10:00 a.m. "The Behavioral Sciences in Medical Theory and Practice"
Edward Stainbrook, M.D., Los Angeles

10:00-10:30 a.m. Visit Exhibits

10:30-11:00 a.m. "Common Psychosomatic Mechanisms—Facts and Fancies"
Bernard I. Lewis, M.D., Palo Alto

11:00-11:30 a.m. "The Psychophysiology of Emotion"
Edward Stainbrook, M.D., Los Angeles

11:30 a.m.-12:00 noon "The Chronic Hyperventilation Syndrome"
Bernard I. Lewis, M.D., Palo Alto

Wednesday P.M., May 12

1:45-2:15 p.m. "The Stresses of Aging"
Alexander Simon, M.D., San Francisco

2:15-2:45 p.m. "Some Observations on Dying Patients"
Carl B. Pollock, M.D., Denver

2:45-3:15 p.m. "The Treatment of the Older Patient"
Alexander Simon, M.D., San Francisco

3:15-3:45 p.m. Visit Exhibits

3:45-5:00 p.m. Panel: "Emotional Problems of Aging and Retirement"
Moderator:
Robert A. Senescu, M.D., Albuquerque
Panelists:
Bernard I. Lewis, M.D.
Carl B. Pollock, M.D.
Alexander Simon, M.D.
Edward Stainbrook, M.D.

6:30-7:30 p.m. Conference Cocktail Party—Courtesy Rocky Mountain Medical Conference
Poolside, La Fonda

7:30 p.m. New Mexico Medical Political Action Committee
Banquet Speaker:
To be announced

Thursday A.M., May 13

Presiding: M. A. Gold, M.D., President, Montana Medical Association

9:00-9:30 a.m. "Hyperbaric Oxygen—Its Uses and Possible Abuse"
William D. Cochran, M.D., Boston

9:30-10:00 a.m. "Pulmonary Reactions to Irritant Gases and Inhaled Particulates"
Hollis G. Boren, M.D., New York

10:00-10:30 a.m. "Smoking and Its Relationship to Bronchitis and Emphysema"
Oscar Auerbach, M.D., East Orange, New Jersey

10:30-11:00 a.m. Recess to Visit Exhibits

11:00 a.m.-12:30 p.m. Panel: "Bronchitis and Emphysema"
Moderator:
Julius L. Wilson, M.D., Santa Fe
Panelists:
Oscar Auerbach, M.D.
Hollis G. Boren, M.D.
Theodore Finley, M.D.
William D. Cochran, M.D
Martin B. Goodwin, M.D., Clovis

Thursday P.M., May 13

Presiding:
Samuel B. Childs, M.D., President, Colorado Medical Society

2:00-2:30 p.m. "Metabolic Aspects of the Pathogenesis of Osteoarthritis"
Alfred J. Bollet, M.D., Charlottesville, Virginia

2:30-3:00 p.m. "Hyperbilirubinemia in Infants"
Audrey K. Brown, M.D., Charlottesville, Virginia

3:00-3:30 p.m. "Age, Hormones and Calcium in the Pathogenesis of Osteoporosis"
Alfred J. Bollet, M.D.

3:30-4:00 p.m. Visit Exhibits

4:00-5:00 p.m. Panel: "Iso-Immune and Auto-Immune Phenomena in Disease"

Moderator:
Richard B. Streeper, M.D., Santa Fe
Panelists:
Brown M. Dobyns, M.D., Cleveland
Alfred J. Bollet, M.D.
Audrey K. Brown, M.D.

6:30-7:30 p.m. Cocktails
Poolside—La Fonda

7:45 p.m. Banquet—Dining Room
Dancing

Friday A.M., May 14

Presiding:
Howard P. Greaves, M.D., President, Wyoming State Medical Society

9:00-10:00 a.m. "The Pathogenesis of Peptic Ulcer"
Lester R. Dragstedt, M.D., Gainesville, Florida

10:00-10:30 a.m. "The Treatment of Nodular Goiters—Thyroid Substitution Therapy or Surgical Exploration"
Brown M. Dobyns, M.D., Cleveland

10:30-11:00 a.m. Visit Exhibits

11:00 a.m.-12:30 p.m. Panel: "Thyroid Nodules"
Moderator:
R. C. Derbyshire, M.D. Santa Fe
Panelists:
Brown M. Dobyns, M.D.
Robert S. Stone, M.D.
Richard M. Angle, M.D.

Friday P.M., May 14

Presiding:
John M. Read, M.D., President, Nevada State Medical Association

2:00-2:30 p.m. "Total Surgical Correction of Tetralogy of Fallot"
Norman E. Shumway, M.D., Palo Alto

2:30-3:00 p.m. "Clinical Renal Heterotransplantation"
Keith Reemtsma, M.D., New Orleans

3:00-3:30 p.m. Visit Exhibits

3:30-4:00 p.m. Alvin J. Ingram, M.D., Memphis

4:00-5:00 p.m. Panel: "Peptic Ulcer"
Moderator:
James Clarke, M.D., Albuquerque
Panelists:
Robert T. Cauthorne, M.D., Albuquerque
Lester R. Dragstedt, M.D.
Keith Reemtsma, M.D.

Friday Evening
Specialty Society Meetings

Saturday A.M., May 15

Presiding:
Robert P. Beaudette, M.D. Raton, President-Elect, New Mexico Medical Society

9:00 a.m.-12:00 noon Panel: "Transplantation"
Moderator:
Solomon Papper, M.D., Albuquerque
Panelists:
Alfred J. Bollet, M.D.
Keith Reemtsma, M.D.
Norman E. Shumway, M.D.

AUXILIARY PROGRAM

Wednesday, May 12

9:00 a.m.-12:00 noon Executive Board Meeting
Tentatively Scheduled House and Garden Tour

12:15-1:30 p.m. Executive Board Luncheon

2:00-4:30 p.m. Workshop

Thursday, May 13

9:00 a.m.-12:00 noon Delegates' Meeting

1:00 p.m. Luncheon
Speaker:
Mrs. R. C. L. Robertson, National Representative

6:30-7:30 p.m. Cocktails
Poolside, La Fonda

7:45 p.m. Banquet—Dining Room
Dancing

Friday, May 14

9:00 a.m.-12:00 noon New Executive Board Meeting

Cataclysmic Deterioration of the Elderly Patient*

Richard M. Angle, B.S., MSc (Med), M.D.** *Santa Fe*

A cataclysm, according to Webster's New International Dictionary (1), while literally referring to a sweeping flood of water, figuratively means an upheaval "likened in its violence to a deluge". The term "cataclysmic deterioration of the elderly patient" is proposed to describe the illness of an elderly patient who is living in equilibrium with one or more chronic diseases when stricken with another acute affliction which seems to trigger a chain reaction of medical catastrophes leading to inevitable death. Frequently the final cause of death is unrelated, or only distantly related, to the condition which initiated the final illness. The tenuous grasp on life held by many older individuals and the ease with which this grasp is broken by a relatively minor illness is well known. The cases included in this category are characterized by an almost chain reactive development of serious complications, each seemingly more severe than the preceding one and each adding impetus to a relentless and often rapid downhill course. Always the physician is one step behind in the race to the finish line and reversal of the direction of the race seems impossible. The following cases are examples of such catastrophes.

Report of Cases

Case #1. M.R. A 70 year old white man entered the hospital on 12/26/55 for investigation of left upper abdominal cramping pain associated with loose stools of three weeks' duration. Three years previously he had undergone a resection of part of the descending colon for carcinoma and there was a history of bronchial asthma and chronic cough. His blood pressure was 120/80 and other pertinent physical findings included dry crepitant rales at both lung bases, moderate tenderness of the left upper quadrant of the abdomen, and a mild coolness of the lower left leg with reduced pulsations in the palpable arteries of both legs.

On the second hospital day a barium enema examination resulted in failure of the barium to pass beyond the splenic flexure although the site of the anastomosis from the previous colon surgery was identified seven cm. distal to the point of the obstruction. In the afternoon of this day he developed pain and cyanosis of his left leg. A surgical consultant advised Papaverine parenterally. During the ensuing night the patient became confused and attempted to get out of bed, sliding onto the floor and landing forcibly on his left leg. He complained of pain in the left knee and ankle which were subsequently adjudged normal by x-ray.

On the third hospital day the barium enema was repeated and barium passed through the constricted area leading to a presumptive diagnosis of carcinoma of the splenic flexure. Because of increasing cyanosis and coldness of the left leg a lumbar sympathetic block was performed.

On the fourth hospital day the patient complained for the first time of pain in the left hip and x-rays revealed a badly comminuted fracture of the neck of the femur. An othopedic consultant placed a Kirsner wire in the ankle and instituted traction because of the precarious circulation of the leg. The lumbar sympathetic block was repeated.

Late in the fourth hospital day the patient com-

*Presented at the Regional Meeting of the American College of Physicians, Albuquerque, December 7, 1963.

**Clinical Associate in Medicine, Univ. of New Mexico School of Medicine.

plained of increasing abdominal pain which by morning of the fifth day was confirmed as being due to a complete intestinal obstruction. Abdominal exploration was performed and an obstruction of the mid-ileum due to adhesions was found and released. The tumor of the splenic flexure was confirmed and a palliative 17 cm. resection of the colon with end-to-end anastomosis was done. The first post-operative night was hectic when the patient became totally irrational and both legs became severely cyanotic. On the morning of the second post-operative day the patient had a transient episode of tachycardia with marked drop in blood pressure but with spontaneous correction. An electrocardiogram showed a left ventricular strain pattern but no other abnormality. Five hours later and about 24 hours post-operatively his pulse became extremely rapid with no detectable blood pressure. An electrocardiogram revealed a ventricular tachycardia. The patient expired 10 minutes later while receiving procaine amide under electrocardiographic control.

Autopsy revealed marked cardiac dilatation and a bifurcation thrombus of the abdominal aorta. The surgical specimen revealed a primary adenocarcinoma of the colon. Death was due to ventricular tachycardia.

COMMENT — In this patient admission for a barium enema set off a chain reaction of catastrophes leading to death in six days. One could almost say he would have fared better had he avoided the physician, but obviously he was on the verge of serious trouble when he entered the hospital.

Case #2. B.H. This 71 year old white woman entered the hospital on 6/27/60 because of severe nausea, vomiting and prostration of 24 hours duration, the onset of which followed by a few hours the eating of leftovers from her own kitchen. Her right breast had been removed radically 4 years previously because of carcinoma. On admission her temperature was 103.2 F. and examination revealed moist crepitant rales of both posterior lung fields and diffuse abdominal tenderness. On admission, the urinalysis was normal and the white blood count was 23,200 WBC/cu. mm. A chest x-ray was interpreted as showing a bilateral bronchopneumonia. On the second hospital day she was semi-stuporous and exhibited nuchal rigidity. A spinal puncture yielded cloudy fluid from which was grown a coagulase-positive hemolytic staphylococcus which was also grown from the urine, blood and stool. The patient responded well to massive antibiotic therapy consisting of erythromycin, penicillin, streptomycin and ristocetin, was afebrile in three days and all medication was stopped after 12 days.

On the 15th day, however, fever recurred and cultures of the urine, blood and stool again yielded a hemolytic staphylococcus which was resistant to all antibiotics previously used. Vancomycin was begun. On the 22nd hospital day the patient developed a left hemiplegia. After 18 days administration of vancomycin was discontinued on the 33rd day because of an increasingly severe rash after each injection and treatment with kanamycin was begun. On the 35th day, two days after vancomycin was stopped and 13 days after the hemiplegia had occurred a harsh systolic apical murmur was first noted. The patient received kanamycin from the 33rd through the 63rd day, during which time the temperature remained normal. The murmur persisted, however. Weekly blood cultures remained sterile and one subsequent spinal fluid culture showed no growth. The patient was slowly recovering from the hemiplegia when on the 83rd day acute pulmonary edema developed and she died in 30 minutes, despite therapy. No autopsy was permitted.

COMMENT — In this case, a woman apparently cured of carcinoma of the breast incurred a severe staphylococcus enteritis, meningitis, pyelonephritis, pneumonitis and endocarditis, presumably as a result of eating contaminated food. Although initial antibiotic therapy seemed adequate, recurrence of the infection occurred with bacterial resistance. Again specific therapy seemed to eradicate the infection but not until after cerebral embolization had occurred and valvular damage had developed, leading to death from acute pulmonary edema. The illness lasted 84 days.

Case #3. T.B. This 76 year old white man had been under treatment for six months for severe pulmonary emphysema and fibrosis and pulmonary heart disease but had discontinued treatment two weeks previously for no apparent reason. He had exhibited severe mental confusion, cyanosis and dyspnea for four hours prior to admission to the hospital on 4/18/60. Initial examination revealed a normal temperature, blood pressure of 120/70

and a pulse of 90. Further examination showed distention of the neck veins in the erect position, increased PA diameter of the chest with diminished breath sounds and minimal basal rales, an apical systolic heart murmur, moderately severe hemorrhoids, symmetrical enlargement of the prostate gland and a trace of edema of the legs. The admission electrocardiogram revealed an incomplete right bundle branch block and was consistent with right ventricular hypertrophy. The chest x-ray showed pulmonary emphysema and fibrosis and was interpreted as showing evidence of pulmonary hypertension.

For the first week the patient showed a good response to diuretic management and correction of his respiratory acidosis. However, on the ninth hospital day he developed diarrhea and abdominal distention and a scout film of the abdomen revealed an extensive paralytic ileus. This was thought to be due to a mesenteric thrombosis. Nasogastric suction was instituted and by the 14th hospital day the abdomen was again soft with no radiographic evidence of ileus.

On the 15th hospital day the patient became extremely irrational and combative, claiming that he was being poisoned, and from this point on refused all food and fluid.

On the 17th hospital day fever and left lower lung rales led to a diagnosis of bronchopneumonia which was confirmed by chest x-rays. Sputum could not be obtained for culture but erythromycin given empirically was effective and by the 22nd day the patient was afebrile. However, he remained irrational and combative and had to be restrained. He continued to refuse all oral medication and liquids and had to be fed parenterally. On the 26th day fever and bilateral lung rales recurred and his wife refused permission for further parenteral treatment. Coma ensued and the patient expired on the 30th hospital day.

Autopsy examination revealed sever pulmonary emphysema and aspiration pneumonia of the right upper lobe. The heart showed slight hypertrophy with coronary artery sclerosis and focal myocardial fibrosis. There was a chronic duodenal ulcer and focal cortical hyperplasia of the adrenal glands. The kidneys showed evidence of arteriosclerosis. The prostate gland revealed several infarcts and, surprisingly, a small well-defined carcinoma. There was no evidence of mesenteric thrombosis.

COMMENT — In this case the patient stopped effective treatment for chronic pulmonary failure with resulting respiratory acidosis, bronchopneumonia, paralytic ileus of unknown cause, and final terminal toxic psychosis and dementia. His final illness lasted 30 days.

The three cases which have been presented have several things in common. All were 70 years of age or older and all had or had had some chronic illness. Two had had previous surgery for cancer and Case #1 had a second previously undiscovered cancer. Likewise, Case #3 had a small but well developed carcinoma of the prostate although this was not felt to have contributed to his final illness. In each case the cause of death was only distantly related to the specific condition which prompted hospitalization. In each instance the course was relentlessly downhill and one complication was quickly followed by a new and more formidable one. In Case #1 the speed of the terminal events was frightening, and although slower in Cases #2 and #3, was equally disastrous. In each case the physician was slightly behind the pace at which deterioration occurred. Despite what appeared to be specific therapy, death resulted.

Every physician who cares for geriatric patients has undoubtedly encountered similar cases. Certainly sudden death in the chronically ill elderly patient is not uncommon and may be caused by relatively minor illnesses. Influenza, for example, while temporarily disabling to the healthy adult, often takes a high toll among those past 60 (2) and those who are suffering from chronic illnesses. The distinguishing feature of those cases for which the term "cataclysmic deterioration" is proposed is the wave-like occurence of complications which in serial fashion leads to death in a period of days or weeks, and the apparent inability of the physician to prevent the succeeding new catastrope. These cases should serve to warn the physician to pay the strictest attention to even seemingly minor illnesses in caring for the geriatric patient. In searching for a term to denote the cases which have been described I consulted my brother, (3) a historian and author, who did not completely approve of the designation but who did state that as a layman, the cases suggested to

him the following quotation from Oliver Wendell Holmes:

Have you heard of the wonderful one hoss shay
That was built in such a logical way
It ran a hundred years to a day? . . .
How it went to pieces all at once, —
All at once and nothing first, —
Just as bubbles do when they burst. (4)

Summary

This report describes three patients in whom the occurrence of an illness upset a state of equilibrium of health and disease. In each case the newly acquired illness triggered a chain reaction of critical complications leading to death in three months. It seems certain that such patients are not rare but no designation has been given the curious succession of catastrophes comprising the final illness, and the apparent inability of the physician to alter the fatal course. These cases should remind the physician of the precarious equilibrium that may exist in the elderly patient living with chronic disease. The term "cataclysmic deterioration" is proposed to describe the occurrence of such tragedies.

Box 2063.

REFERENCES

1. Webster's New International Dictionary of the English Language, Ed. 2, Springfield, Mass, G. & C. Merriam Co., 1958, p 420.
2. Harrison, T.R.: Principles of Internal Medicine, Ed. 4, New York, McGram-Hill Book Company, Inc., 1962 p 1125.
3. Angle, Paul M.: Personal communication to the author.
4. Holmes, Oliver Wendell: The One Hoss Shay, Boston & New York, Houghton, Mifflin & Co., 1892.

Dr. Basom Heads American Fracture Ass'n.

Dr. W. Compere Basom of El Paso, who has been Vice-President of the American Fracture Association for several years, has been elected President of the organization for a three-year term.



Dr. Basom

Dr. Basom is making plans for this year's meeting of the Association this Fall in Las Vegas, Nevada, and for the 1966 session, which will be held in Caracas, Venezuela. The Association has a policy of promoting friendship and scientific knowledge among physicians in North, Central and South America.

Dr. Basom is President of the Texas Division of the International College of Surgeons and presided at the Southwestern Regional Meeting of the College in Houston, March 4-6.

He received his M.D. Degree from Baylor Medical School after completing pre-medical work at Southern Methodist University. Following two years of internship, he did three years of Postgraduate work as a Fellow in Orthopaedic Surgery at the Mayo Foundation and obtained a M.S. Degree in Orthopaedic Surgery from the University of Minnesota. He has been in practice in El Paso with Dr. Louis W. Breck and later Dr. Morton H. Leonard and The El Paso Orthopaedic Surgery Group since 1941 with the exception of four years in the U. S. Medical Corps at William Beaumont General Hospital during World War II.

Medical Education for the Personal Physician*

Merlin K. DuVal, M.D. *Tucson***

Since this conference is attuned to the subject of Graduate Medical Education in General and Family Practice, you may have found it disappointing to note that there is one title which makes no reference to graduate education. This is not the fault of your program committee — it is exclusively my doing — and it represents a concession on their part. However, I suspect that it is not the first time you have expected steak only to receive hamburger.

My new assignment, in the State of Arizona, has required that I become as thoughtful as possible about education for the practice of medicine. And, while graduate medical education will become of great concern to us later, I am confident that these programs will evolve successfully as long as we are particularly careful to define what it is we expect them to accomplish. Here, then, is the difficulty. In the field of general practice, none of us is sure what it is we want our programs to accomplish. I would like to start by considering this phase of the problem with you.

Decline of New G.P.'s

There seems to be no doubt whatsoever that the number of physicians who are entering the general practice of medicine is on the decrease. And while there are honest differences of opinion as to why this is happening, there is complete agreement on some of the factors which are playing a role. Many of these are related to our schools of medicine and to our system of dispensing medical education. For example, the post-Flexnerian emphasis on the basic science disciplines and on university orientation for medical education, has had an effect which can be compared to that which follows the fertilization of a lawn: everything that has been planted, grows. As a result, each of our basic disciplines has become an enormous field in itself, and our medical school curriculum reflects this separation by a lack of cohesion and interrelationship between these disciplines. More than that, the enormous investments in medical research have so expanded the individual fields that subspecialization has appeared as the natural consequence. I am afraid that this has fostered a type of "detachment" among those of us who are involved in the academic side of medicine, a detachment from the outside world, the general public we serve, and, most regrettably, from our own professional colleagues who are out in practice. If this sense of detachment is as prevalent among the speciality-oriented members of our medical school faculties as I am suggesting that it is, then it should not come as any great surprise that the environment for the medical student will not be geared toward the general practice of medicine.

Important Developments

At the same time, several important things have been happening among the practitioners themselves; things which are not related to the medical schools. For example, there is strong evidence that general practice has not proved to be as satisfying a career as we might wish to think it is. The number who leave general practice and return for additional post-graduate training in a speciality area is substantial. Studies now completed in two states reveal that slightly less than one-third of all the physicians who are practicing in those states have taken specialty training. Presumably, this would leave more than two-thirds who should be doing general practice. Surprisingly enough, only three out of 10 listed themselves as general practitioners. All of the others designated themselves as having added a speciality to their practice. Is *this* the group which is eager to see young men trained to do something they are not willing to do? It may be pertinent to recall here that the American Specialty Boards were founded by men who limited their practices, and that as recently as three years ago the American Medical Association received a petition to approve another new speciality board: The Board of Abdominal Surgery. In a slightly different

*Presented at the meeting of the American Academy of General Practice, Hartford, Connecticut, January 30, 1965.
**Dean, College of Medicine, The University of Arizona, Tucson.

vein, some of my closest and respected friends in the field of general practice have told me that men coming into their communities recently, presumably to enter the general practice of medicine, are often unwilling to accept their share of the coverage of the local hospital emergency room; they hold office hours "by appointment only" and not infrequently have unlisted telephone numbers. Is this the image of general practice you would have us forward to the student body?

Three Reasons

Perhaps the situation I am describing is not so much a "fault" as it is a phenomenon; a phenomenon which, by the way, will get worse rather than better if we persist in our efforts to reorient and adapt today's medical science to the goal of increasing the supply of yesterday's general practitioner. In fact, if we do continue in this direction we may even hasten the disappearance of this general practitioner for three reasons: first, since we are continuing to produce new knowledge at a formidable rate, each newly graduated physician will be more knowledgeable, in many academic areas, than the man who is actually out there on the firing line. This will inevitably make the latter physician feel more and more "left out" and "left behind" and the attractiveness of his practice will diminish. Second, in a specialty-conscious society (as more specialists become available) the hospital privileges, prestige, and community standing of the general practitioner will be increasingly threatened. Third, the spectacular growth of ancillary health agencies, in both private and governmental sectors (coordination between which is atrocious by its absence), represents a very important source of competition for services to the patient, all of which will detract from the importance of the general practitioner.

Since medical schools are themselves the focal point of new knowledge, and are continuing to add to this body of knowledge, it seems safe to assume that the influence of the specialist will continue to be represented on their staffs. Furthermore, as long as our medical curriculum retains its Flexnerian rigidity, we need not expect this cycle to break. However, before you leap to the conclusion that all we have to do to correct this situation is to change the medical school curriculum and to introduce departments of general practice within medical school faculties, let me remind you that there is already ample proof that such steps, having been taken, have had no substantial impact on the production of family doctors thus far.

Ultimate Solution

The ultimate solution to this problem is going to require something quite different. Let me be specific: first, no solution is going to be possible until we concede that specialization is not only here to stay, but should be recognized as a sound and important step in social progress whether it be in medicine, agriculture, law or, for that matter, in football. In other words, a solution which tries to keep down the influence of the specialist is doomed to fail. Second, it is completely unreasonable for us to persist in our efforts to train a general practitioner to fit the same mold that we know he fitted in the past. That this mold has stood the test of time for several decades is no more persuasive or logical than is the argument that the internal combustion engine, which has also stood the test of time, should be adapted to the requirements of space travel. Therefore, any effort which is geared to the continued production of the general practitioner of the past is likewise doomed to failure. Third, we had better wake up pretty quickly and realize that the market place for our medical services continues to reveal a demand for both the specialist and the family, or personal, physician. As long as that demand persists, we would be terribly short sighted not to make an effort to satisfy it since, if we don't, you can be sure that it will be satisfied by others.

If the basic theme of these postulates is correct, then it can serve as a framework within which the specific solution to our problem becomes possible. The three-point solution which I would like to offer you for your consideration today is based on these postulates and may be summarized as follows.

Summary

First, I think it is essential that the future of general practice be redefined in terms of the health needs and requirements of our society, rather than in terms of the preferences of the individual physician. In this sense, and in response to my interpretation of what our public is looking for, I visualize the generalist of the future as assuming three important roles: dispensing continuous medical treatment for the great majority of the specific ills of his patient clientele; serving as an intensely per-

sonal advisor and confidant to his patients in all matters related to their physical and emotional well-being; and as the key consultant in a determination of what other resources should be mobilized on behalf of his patient. Note that I am not describing a personal physician as a "second-class doctor" but rather that I am describing a position of the utmost responsibility and trust — which is precisely what your patient is seeking even though he is surrounded by physicians, a choice of specialists, laboratory and medical technicians, nurses, private and governmental health, welfare and social agencies — all of which are eager to satisfy his wants. Do we need reminding that the health industry now employs more persons than any other industry in the United States except for agriculture, and that these people will fill the vacuum you leave — if you choose to leave it? In this role, a personal physician will be very much of a specialist himself. He will have to have many attributes and capacities, as well as substantial knowledge, which most of the other specialists will not have.

Second, if we can agree that society does have a need for personal physicians, then it follows that we must reorient our undergraduate and graduate medical curricula to stimulate the production of such physicians, and then keep them up to date. This doesn't strike me as particularly difficult. Its only real requirement is that we first make a genuine effort to determine what kind of education and training the personal physician will need, and then distribute that knowledge so that it can be geared to his anticipated requirements. We can expect this knowledge to be heavy in the field of internal medicine, pediatrics, obstetrics, preventive medicine, behavioral science, office surgery, the emergency, and all of the health resources. Meanwhile, speciality emphasis would be shifted toward the graduate years, thereby greatly diminishing the influence on the student of the sub-speciality departments. Instruction within the medical school would be interdisciplinary in character, but there would be a shift in the focus of medical education away from the patient who is supine in a "teaching hospital" and toward the patient who is ambulatory, and preferably outside the hospital. After graduation, continuing education takes over. Unfortunately, I am afraid that our medical schools have placed a disproportionate emphasis on teaching programs for the undergraduate student in the past, as compared to programs for the man in practice. A medical school will never rise to its true responsibility until it accepts its larger role as the instrument through which medical knowledge is transmitted to all members of the profession, at whatever stage of development or maturity they may be. The assumption of this role by the medical school, if accomplished simultaneously with the redefinition of general practice, and its attendant curriculum change, will have a welding effect within our profession the like of which we have not seen in many decades.

It is this later aspect which brings up the third and last step which, as a profession, we must take together. I refer to the amalgamation of the medical profession into a solidified unit in which each segment recognizes and acknowledges the merit, contributions, and worth of the others with which it is interdependent. This is the only route that will permit us to present a successful professional posture to those with whom we have to deal, be they insurance carriers, federal and state governments, or our general public. Only by this route will we be construed as eligible to reassert the initiative and maintain our position of leadership in health-related affairs. But this could be a tall order, and may require a type of firmness and discipline even beyond that which we now have within our profession. We may find it necessary, for example, to license and qualify ourselves in different categories, thereby following the leadership already demonstrated to society by other health professions such as nursing and osteopathy. But how much more preferable that we do this by ourselves than have it done to us, indirectly, through the control of hospital privileges, third-party payments, etc.

If men of our profession have dignity, and are unafraid, then the required discipline and respect will follow as a matter of course. The steps I have offered for your consideration represent a route by which I believe our profession can achieve the stature and self-confidence it requires.



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Benactyzine hydrochloride—Benactyzine hydrochloride, particularly in high dosage, may produce dizziness, thought-blocking, a sense of depersonalization, aggravation of anxiety or disturbance of sleep patterns, and a subjective feeling of muscle relaxation, as well as anticholinergic effects such as blurred vision, dryness of mouth, or failure of visual accommodation. Other reported side effects have included gastric distress, allergic response, ataxia, and euphoria.

Meprobamate—Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

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Coming Meetings

17th Annual Scientific Assembly of the American Academy of General Practice, Civic Auditorium, San Francisco, April 12-16, 1965.

Third National ISA (Instrument Society of America) Biochemical Sciences Instrumentation Symposium, Statler Hotel, Dallas, April 19-21, 1965.

Medical-Surgical Seminar on Pulmonary Diseases, Wm. Beaumont General Hospital, El Paso, April 20, 1965.

23rd Annual Meeting, U.S.-Mexico Border Public Health Ass'n., Los Angeles, April 26-29, 1965.

83rd Annual Meeting of the New Mexico Medical Society and 12th Biennial Meeting of the Rocky Mountain Medical Conference, La Fonda, Santa Fe, May 9-15, 1965.

Eighth Annual Ruidoso Summer Clinic, sponsored by the New Mexico Chapter of American Academy of General Practice, Ruidoso, N. M., July 19-22, 1965. Headquarters: Chaparral Motel.

62nd Annual Meeting of the Westen Association of Railway Surgeons, Alameda Hotel, Avenida Juarez 50, Mexico City, October 27-30, 1965.

15th Annual Meeting of the Southwest Obstetrical and Gynecological Society, Arizona Inn, Tucson, Oct. 28-30, 1965.

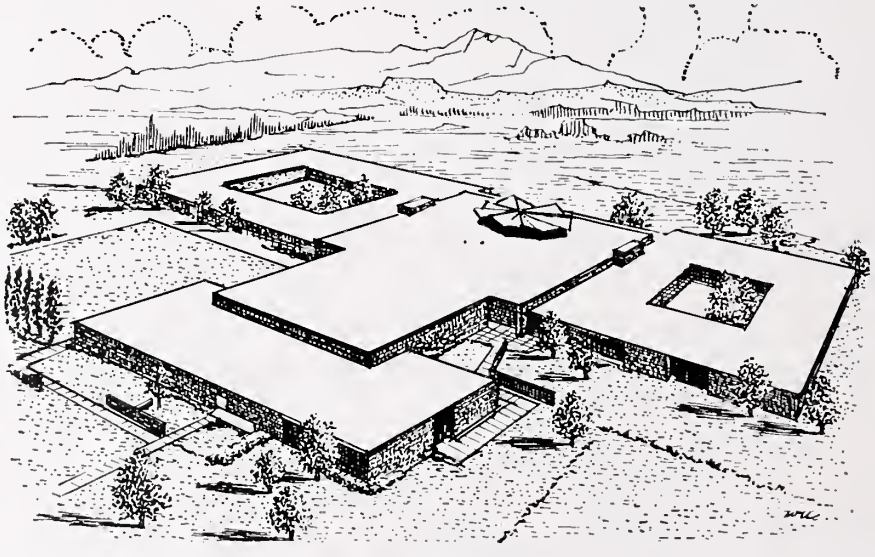
47th Annual Meeting, Southwestern Medical Association, Sheraton Motor Inn, El Paso, November 4-6, 1965.



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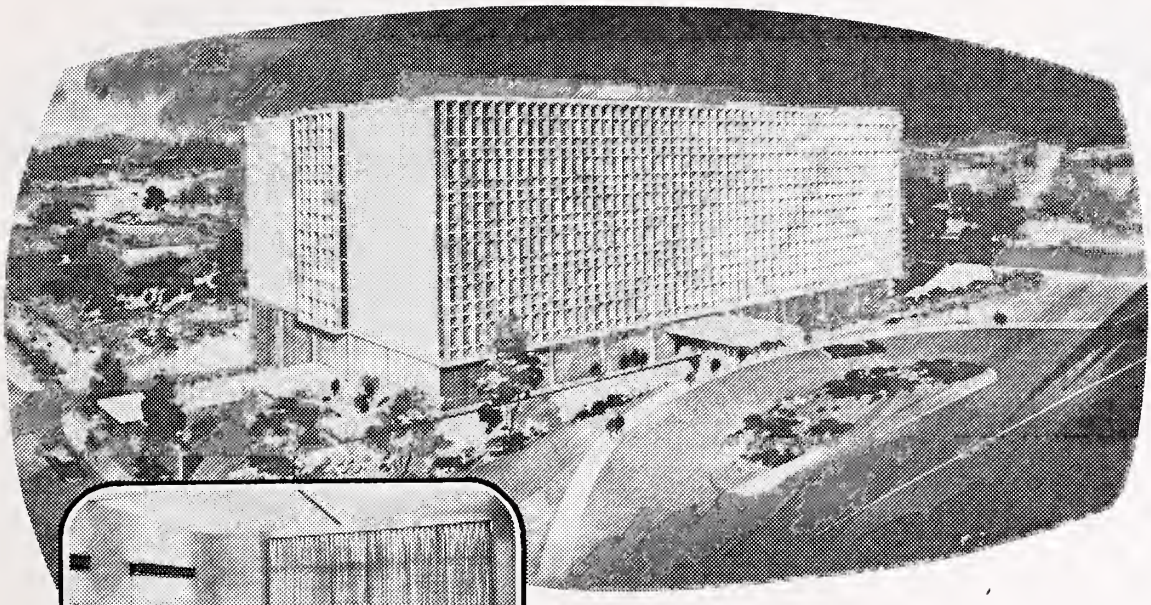


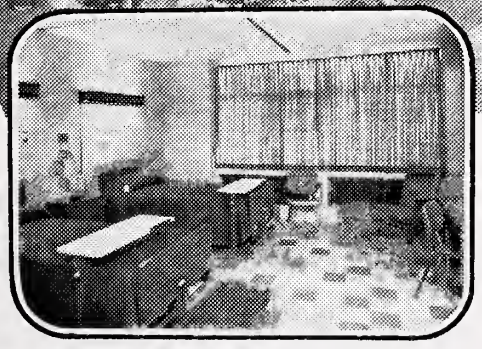
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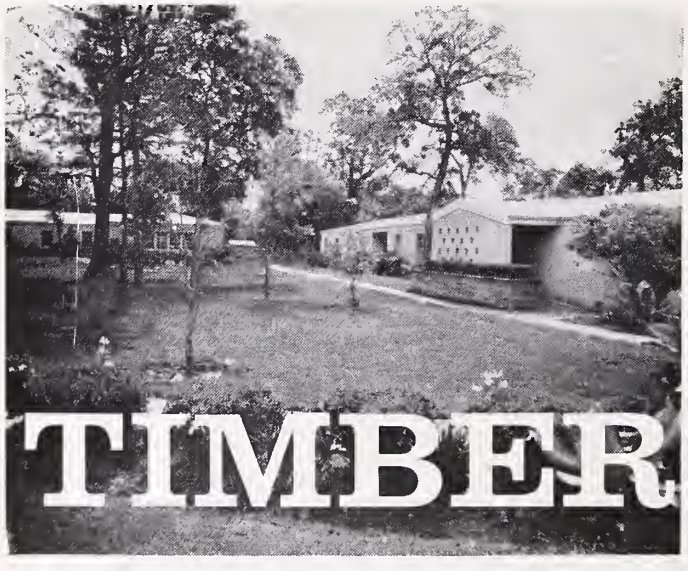
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DOSAGE: One or two tablets four times a day. After remission of symptoms dosage should be reduced to the minimum maintenance level.

SIDE EFFECTS: Nausea, GI upset, or mild salicylism may rarely occur. Symptoms of hypercorticoidism dictate reduction of dosage of Arthralgen-PR.

PRECAUTION: Reduction in dosage of Arthralgen-PR given over a long period should be gradual, never abrupt.

CONTRAINDICATIONS: Hypersensitivity to any ingredient.

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SUPPLY: Arthralgen (white, scored) and Arthralgen-PR (yellow, scored) tablets are available in bottles of 100 and 500.

REF: 1. Boreus & Sandberg, ACTA. PHYSIOL. SCAND., 28:266, 1953. 2. Cohen, et al.: J.A.M.A., 165:225, 1957.

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1. Griffith, R.S., and Black, H.R.: Current Ther. Res., *6*: 253, 1964.

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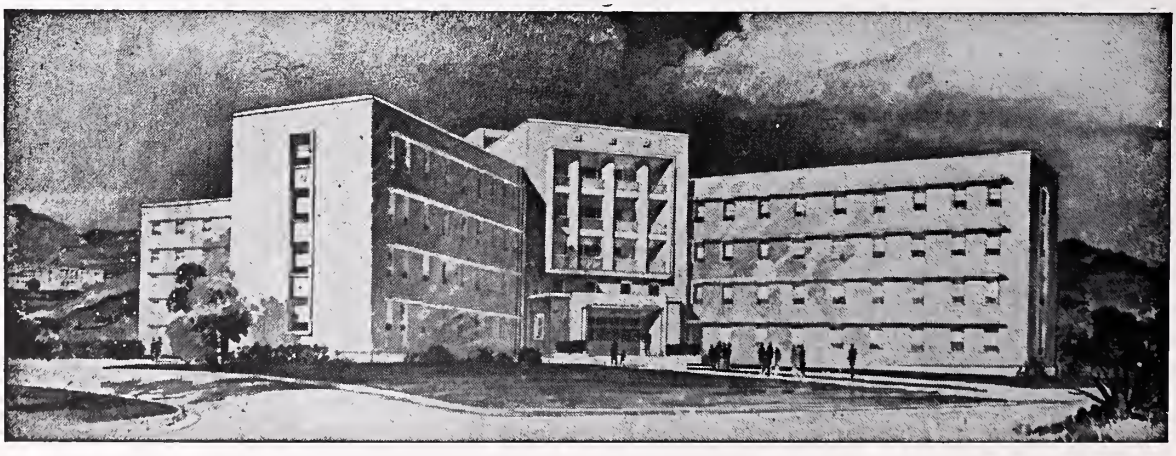
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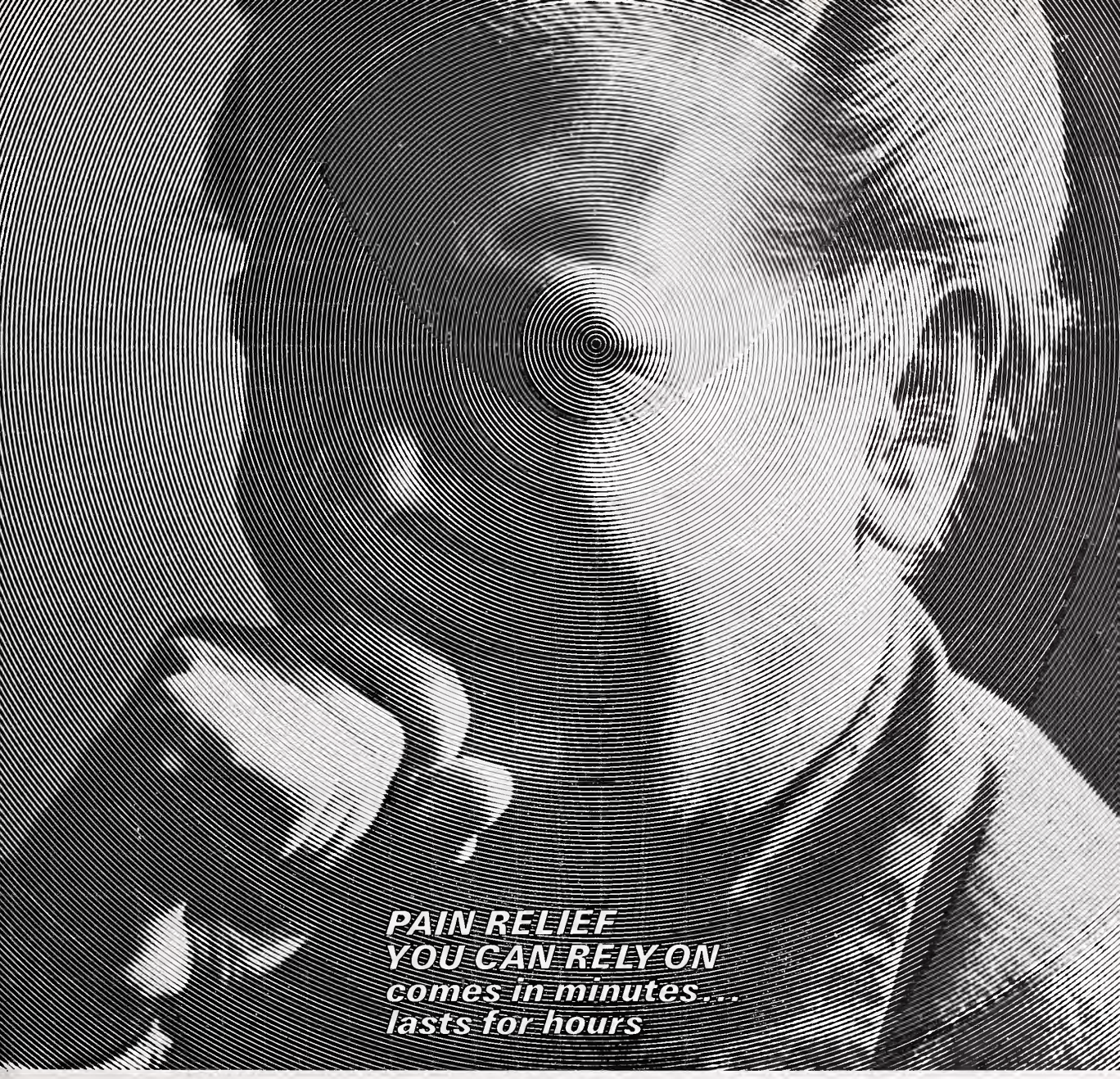
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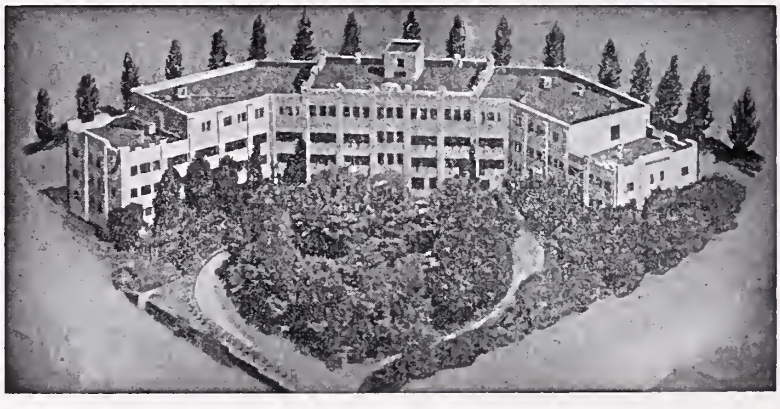
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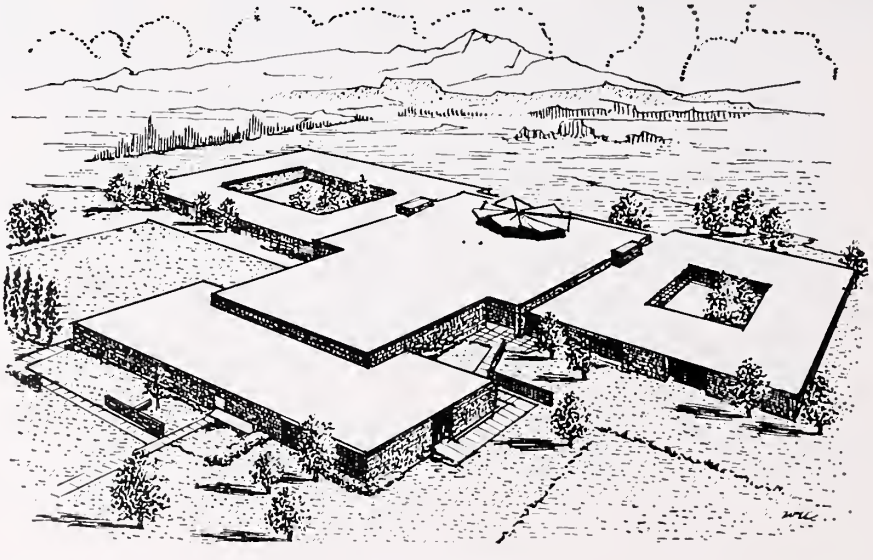
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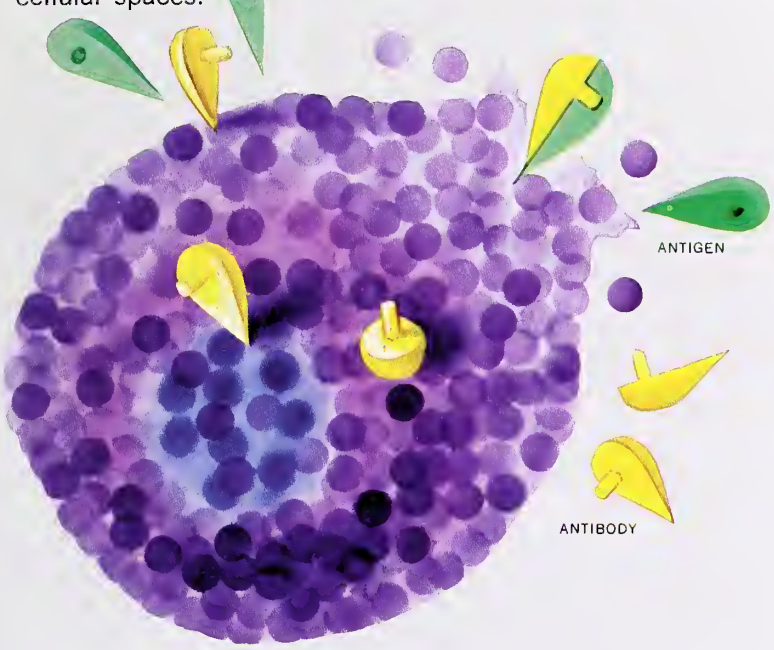
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*Schiller, I.W. and Lowell, F.C.: New England J. Med. *261*:478, 1959.

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REFERENCES: 1. Soto-Hall, R.: Med. Sci. *14*:23, 1963. 2. McCarrol, H.R.: Paper read at the Annual Meeting of the American Medical Association, Atlantic City, June 16-20, 1963. See Medical News: J.A.M.A. *185*:39 (July 13), 1963. 3. Gordon, E.J.: Med. World News *5*:54, 1964. 4. Cozen, L.: GP *26*:82, 1962. 5. Larson, C.B.: Postgrad. Med. *26*:142, 1959. 6. Forsyth, H.F.: J.A.M.A. *167*:163, 1958. 7. Weiss, M., and Weiss, S.: J. Amer. Osteopath. Ass. *62*:142, 1962. 8. Rowe, M.L.: J. Occup. Med. *2*:219, 1960.

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Neurological Changes Common to Advanced Age*

Arnold H. Greenhouse, M.D.**, *Albuquerque*

Elderly patients are subject to several neurological disorders not shared by younger individuals. Certain abnormalities, such as distal loss of vibratory sense, decrease or absence of the ankle jerks, and difficulties of ocular convergence and elevation are so common that they lose special significance. Tremulousness, rigidity and gait disturbances are frequent;[1] their etiology is not understood, nor are effective treatment methods known. Mental changes are the most distressing and common of the neurological dysfunctions that accompany aging. Unfortunately, intellectual disturbances are usually blamed on cerebral arteriosclerosis or senility, despite the fact that there may be potentially reversible causes. This discussion will focus on altered mentation, because it is such a serious and poorly understood aspect of geriatric practice.

Cerebral Physiology of Aging

An inquiry into the psysiology of the aging brain helps surprisingly little in the understanding of mental deterioration. Cerebral oxygen utilization, which stays at a remarkably constant 3.5 ml./100 gm. of brain tissue/min. under a wide variety of conditions, is usually not reduced in normal or demented elderly people.[2] However, in dementia associated with significant cerebral arteriosclerosis, values drop to an average of 2.7 ml./100 gm. of brain tissue/min.[3] Even levels of 2.0 ml./100 gm. of brain tissue/min. are found in various states of coma. It is generally agreed that significant reductions of cerebral oxygen utilization in the elderly are due to arteriosclerotic vascular disease.[3]

Normal values for cerebral blood flow are 50-60 cc./100 gm. of brain tissue/min. Great variations have been reported in aging, depending on the method employed. Most studies have shown a gradual, progressive fall of cerebral blood flow beginning in the fourth or fifth decade.[2] Unfortunately, many of these studies of "normal patients" actually employed arteriosclerotic subjects. Recent investigations indicate that cerebral blood flow in aged persons with good physical and mental health does not decrease significantly.[4] Only in association with definite cerebral arteriosclerosis is there a diminution of cerebral blood flow. Normal values are obtained in essential hypertension and senile dementia.[2, 4]

The only constant cerebral metabolic change found in old age is in glucose utilization.[3, 4] Even in elderly patients with completely normal mentation there are significant reductions from the normal of 5 mg. of glucose/100 gm. of brain tissue/min. The reason for this reduction is not known, although it is speculated that it may be associated with alterations in enzymatic function.

There is some dispute about the status of the EEG in aging. There seems to be an increase in fast activity, some lowering of voltage and slowing of the normal alpha frequencies.[5] Slow rhythms, at less than the normal alpha rates, are not usually seen in older persons, although some investigators claim that bitemporal slowing in normal aged persons indicates asymptomatic cerebral vascular disease. Generalized slow activity occurs in the senile and pre-senile dementias, yet patients with these disorders may have normal records. A normal EEG can be of diagnostic help in the evaluation of dementia, since it might suggest a potentially reversible disorder. Marked focal slow activity

*Presented at the Fourth Annual New Mexico Neuropsychiatric Seminar, Clovis, New Mexico, October 2, 1964.

**Assistant Professor of Medicine (Neurology) and Chief, Neurology Section, University of New Mexico School of Medicine, Albuquerque, New Mexico.

would indicate a focal lesion such as a tumor, infarct or abscess.

CNS Pathology of Old Age

Cerebral arteriosclerosis is commonly found at autopsy in the aged, but its significance in relation to dementia is difficult to assess. Severe atherosclerosis may occur in patients whose mentation was good, whereas markedly demented persons frequently have normal cerebral vessels. Cerebral vascular disease is usually limited to older persons since it generally begins 10 to 15 years later than the same process in the aorta and coronary arteries. Exceptions to this rule are found; occasionally strokes are seen in the young and often very old patients have normal intracranial vessels. There is very little difference in the sex incidence of cerebral arteriosclerosis, unlike the situation in coronary disease where males predominate. However, when strokes do occur in the younger age groups they are much more frequent in men.

Gross atrophy of the brain is common in old age. The convolutions become narrow and the sulci widen, particularly in the frontal and temporal lobes. The leptomeninges often thicken, become adherent and an excess of CSF collects in the leptomeningeal spaces. The ventricles dilate and white matter shrinkage is obvious on cut sections of the brain. The space between skull and brain doubles in size between ages 30 to 70. This increased space is of clinical importance, since mass lesions such as a tumor or subdural hematoma can grow to a considerable size before causing symptoms. The total brain weight declines from an average of 1400 gm. at age 20 to 1170 gm. at age 80.

A wide variety of histologic changes are seen in the aging brain. Cerebral neurones appear swollen, their cytoplasm pales, Nissl substance is reduced, the nucleus becomes more basophilic and a lipochrome pigment is deposited. There seems to be a decrease in the total number of cerebral cortical neurones, but the degree of loss is difficult to ascertain and it is by no means always proportional to the patient's age. Astrocytes become more fibrous and accumulate pigment. There is a general tendency toward an increase of glial fibers throughout the brain. Cerebral blood vessels thicken, become less pliable and the media undergoes fibrosis.

Senile plaques and neurofibrillary degeneration are two interesting structures, both containing amyloid, that may occur in the brain of advanced years. Their demonstration requires silver impregnation techniques, their origin is obscure and they are of unknown significance. Senile plaques are argyrophilic masses of varying size, found in the cerebral cortex and most frequent in the frontal and temporal lobes. They are generally associated with pre-senile and senile dementia, yet they can occur in elderly persons who have normal mentation. Neurofibrillary degeneration is a thickening and contortion of fibrillary structures within the neuronal cytoplasm. This change usually appears only in pre-senile and senile dementia.

Organic Mental States In The Elderly

The most important differentiation to be made in the elderly with problems of mentation is between the "confusional states" which are characterized by gross disturbances of consciousness and the "organic dementias" in which impairment of memory and intellect predominate. A third group consists of patients with focal cerebral disease who may have symptoms of both organic dementia and confusion.

Confusional States (Acute Brain Syndrome): Mental confusion must be distinguished from dementia and its accompanying impairment of memory. Dementia implies an irreversibility not necessarily present in confusion. Confused patients manifest disturbances in contacts with reality, altered awareness of identity and abnormal relations to environment. Symptoms include restlessness, bewilderment, distractability, disorientation, fearfulness, hallucinations, delirium, nocturnal wandering, perseveration, defective perception and inability to register and recall. Obviously, it may be difficult, or impossible, to test confused patients for impaired memory and intellect, yet the very presence of the above symptoms should serve to suggest that the patient may not have an organic dementia.

Confusion, which commonly complicates many diseases in the elderly, is indicative of primary dysfunction outside of the nervous system. Thus, confusion is best considered as a symptom, which should alert the physician that the cause is to be found in extra-cerebral disease. Elderly persons seem particularly prone to the development of confusion because their so-called "brain reserve" (the ability of the brain to withstand various toxic

insults and metabolic deprivations) seems impaired, probably because of structural changes, particularly the neuronal loss previously described. The treatment and prognosis for recovery in confusion are that of its underlying etiology. The possible causes are innumerable and a partial list includes heart failure, pneumonia, emphysema, hepatic failure, gastrointestinal bleeding, diarrhea, pancreatitis, uremia, myxedema, hypoglycemia, fluid and electrolyte disturbances and the use of many drugs such as opiates, sedatives, steroids, diuretics, etc. Almost any systemic disturbance may produce confusion. Organic brain disease should not be overlooked as a potential mechanism; this is particularly true of subdural hematomas, but seizures, small cerebral infarcts, head injuries and certain tumors may be responsible. The demented individual may readily become confused for any of the above reasons, but it is important to remember that the two conditions are not identical.

The Organic Dementias: Dementia may be defined as an irreversible loss of intellectual function. With advancing years most persons undergo some decline in mental abilities which accompanies physical senescence. Memory, intellect, emotional control and range of interests tend to decline. It would be conceivable to diagnose dementia in almost all elderly individuals, but in practical terms such an attitude is not sensible. Yet, it can be difficult to draw a sharp line between mental deterioration and "normal old age". Many mildly demented persons can, through habit and training, manage acceptably in their accustomed surroundings, yet they have considerable difficulty in a strange environment such as a hospital ward.

Early signs of dementia, which may be difficult to detect, include circumstanciality, difficulty in employing abstractions and self-criticism. As the disorder progresses, patients usually demonstrate reduced learning capacity, emotional instability, defective judgment, decreased spontaneity and creativity, poverty of ideas, reduced initiative, and restricted interests. The primary features of dementia are disturbed memory and poor retention. Memory for recent events, which is more profoundly affected than remote memory, can be tested by asking the patient to recall names, addresses, a series of numbers, etc. Certain capacities, such as vocabulary, factual information, and the ability to handle common social situations are usually retained until late in the course of a dementing disease.

External stimulation is extremely important in determining the course and symptoms of dementia. A mildly demented patient does poorly when deprived of interesting environmental stimuli and activity, perhaps accounting for the rapid downhill course which many such persons follow after retirement. Sensory deprivation, even in the young, may lead to bizarre behavior, and the old person, with cerebral neuronal loss and often impaired vision and hearing, is particularly subject to severe difficulties, especially at night or when sedated.

The Causes of Organic Dementia

Many CNS disorders can lead to organic dementia. This outline will list only the major causes, and will not attempt to deal in detail with aspects such as clinical findings and management which are described in standard texts and other sources.

I. Degenerative Disease: The term degenerative, although hallowed by tradition, is unfortunate since it implies a hopeless and incurable condition. Presumably many of these disorders will be defined in the future as to basic mechanisms and treatment.

A. Pre-senile Dementias: Under this heading are included Pick's and Alzheimer's diseases, which are practically indistinguishable clinically and to some extent pathologically. In both conditions there is gross cerebral atrophy, although in Pick's disease the process is most evident in frontal and temporal lobes. Alzheimer's disease is characterized histologically by senile plaques, neurofibrillary degeneration and moderate gliosis. In Pick's disease there are striking neuronal loss, swollen and degenerating "Pick's cells", glial proliferation and occasionally, despite statements to the contrary, senile plaques and neurofibrillary changes. Signs and symptoms in these disorders are similar and include an onset usually between ages 40 and 60, progressive dementia with severe memory loss, seizures and focal neurological signs. The average duration is 2-5 years. The EEG shows slow wave abnormalities and the pneumoencephalogram demonstrates diffuse atrophy. CSF examination is normal.

B. Senile Dementia: This term is reserved for organic dementias occuring past age 65 not due

to cerebral arteriosclerosis, CNS syphilis or other established conditions. Although these patients are too old for the diagnosis of pre-senile dementia, it is not easy to differentiate the two disorders either on the basis of clinical findings or pathology. Many authorities believe that these entities are manifestations of the same process occurring at different ages, although this view is not universally held. The symptoms in senile dementia are those of an organic dementia. Neurological examination may reveal brisk reflexes but focal abnormalities are usually absent and generally there are no important physical findings.

C. Jakob-Creutzfeldt Disease: This is a rare disease, appearing mainly in the fifth and sixth decade, with rapidly progessive dementia, rigidity, tremor, visual loss and myoclonic jerks. Death occurs in six to 12 months. Pathologically there is diffuse cerebral neuronal degeneration and intense gliosis, but senile plaques and neurofibrillary changes are not seen.

D. Huntington's Chorea: In this disease with autosomal dominant inheritance, chorea and dementia are the outsanding clinical manifestations. Profound shrinkage of the corpus striatum and cortical neuronal loss are found in the brain.

E. Neuronal Abiotrophy: Dementia may be a prominent part of the familial group of diseases in which Freidreich's Ataxia and Charcot-Marie-Tooth disease are prototypes.

II. **Vascular Disease:** Under the heading of vascular diseases causing dementia both hypertensive encephalopathy and arteriosclerotic dementia may be included. The former disorder is rare but the latter is fairly common. However, arteriosclerotic dementia is too frequently and incorrectly diagnosed. In order for this diagnosis to be valid there must be definite focal or diffuse neurological findings such as pathologically brisk reflexes, extensor-plantar signs, so-called "pseudobulbar involvement", hemiparesis, etc. A history of previous strokes is helpful. Many patients said to have dementia on the basis of cerebral arteriosclerosis are actually suffering from senility, confusional states and other disorders.

III. **Infectious Diseases:** Paresis, formerly a major cause of intellectual deterioration, has not yet disappeared and many cases will be missed if it is not considered. In order to establish this diagnosis there must be a "positive" spinal fluid with a reactive serology, first zone colloidal gold curve, elevated protein and increased cell count. Dementia can occur in other infectious diseases of the nervous system, including brain abscess, meningitis and encephalitis. In the latter group, herpes simplex encephalitis is particularly important since it often results in a severe and specific dementia closely resembling Korsakoff's psychosis.

IV. **Neoplasms:** Dementia may be the only manifestation of frontal lobe and posterior fossa brain tumors. Severe mental disturbances occasionally develop in patients with extra-cerebral neoplasms who do not have cerebral metastases. In these cases it is presumed that the dementia is of "toxic" origin. It is important to remember that air studies or arteriograms should be performed in all patients with dementia of unknown cause to rule out a neoplasm or other focal lesion.

V. **Deficiency Diseases:** Dementia is common in pellagra, pernicious anemia, Korsakoff's psychosis and Wernicke's encephalopathy. All of these conditions are potentially reversible by the proper vitamin therapy. Thyroid deficiency may develop insidiously in the elderly and severe intellectual loss often results.

VI. **Trauma:** Diffuse cerebral atrophy can occur after head injury, particularly in the elderly who tolerate cranial trauma poorly. It is well known that even relatively minor head injuries cause progressive memory loss in old persons. A form of this disorder is so-called "dementia pugilistica", which frequently develops in boxers years after they cease active fighting. A possible explanation is that repeated head trauma leads to cerebral neuronal injury, and in later years, as the progressive cortical cellular loss of aging develops, a point of cerebral decompensation is reached.

Summary

Of the wide variety of neurological disorders which may be associated with aging, mental changes are the most distressing and common. There are no constant alterations of cerebral physiology found in demented patients to explain their deficit. Gross cerebral atrophy, neuronal loss, senile plaques and neurofibrillary changes can occur, but these findings are by no means specific or seen in all cases. It is particularly important to differentiate organic dementia from the confusional states. The former are characterized by memory impairment, while in the latter there are

gross disturbances of consciousness. The confusional states, which are symptomatic of extracerebral disease, are caused by a wide variety of systemic disorders; they are potentially reversible. The organic dementias may be due to so-called pre-senile and senile degeneration, as well as cerebral arteriosclerosis, infectious processes, brain tumors, various deficiency states and head trauma. Quite often mental deterioration is ascribed to cerebral arteriosclerosis, a diagnosis which is relatively uncommon and should not be made unless there are definite neurological findings.

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Complete Program
Eighth Annual
Ruidoso Summer Clinic

Major topics such as early diagnosis, psychosomatic problems, extremes of life, and vague and misleading signs and symptoms on a program provided by the University of Kansas School of Medicine and talks by Dr. Amos N. Johnson, AAGP President, and Dr. Reginald H. Fitz, Dean of the University of New Mexico School of Medicine, are among features of the Eighth Annual Ruidoso Summer Clinic in Ruidoso, New Mexico, July 19-22, 1965. The meeting is sponsored by the New Mexico Chapter of the AAGP and has as its theme, "Modern Diagnosis".

Members of the Faculty speaking at the meeting have been provided by Dr. Jesse D. Rising, Chairman of Postgraduate Medical Education at the University of Kansas Medical Center. Dr. Herschel L. Douglas, Lovington, Secretary-Treasuer of the N. M. AAGP, is in charge of arrangements for the meeting, which will have its headquarters at the Chaparral Motel. Officers of the New Mexico Chapter are Dr. Bram Vanderstok, Ruidoso, President, Dr. James A. Koch, Albuquerque, President-Elect, Dr. Paul Feil, Deming, Vice-President, and Dr. Douglas.

The popular summer session, high in the pines of the Sacramento Mountains, with afternoons open for a variety of recreation, annually draws physicians from Texas and other nearby states as well as from throughout the nation.

Dr. Johnson and Dr. Fitz will speak at the annual banquet, July 21. Dr. Johnson is a general practitioner from Garland, North Carolina, and is serving as President of the AAGP this year. He has been President of the North Carolina AAGP and is a Past President of the Medical Society of the State of North Carolina. He has been President of the N. C. State Board of Medical Examiners, and is a Past President of the Sampson County Medical Society and the Third District Medical Society. Dr. Johnson is a member of the AMA Ad Hoc Committee to Study Training for Family Practice. For the last three years he was Advisor to the Sears Roebuck Medical Foundation. He was appointed by the Governor to the Hospital Board of Control in 1948 and to the Commission on Court Reform in 1956.

Dr. Fitz, in addition to being Dean of the N. M. Medical School, is Professor of Medicine at the School, is Consultant in Internal Medicine at the VA Hospital in Albuquerque, and Chairman of the Dean's Committee at the Albuquerque VA Hospital. The New Mexico Medical School offers a two-year course and was opened last Fall.

Faculty members at the Ruidoso Clinic will be: Dr. Jack M. Zimmerman, Consultant in Sur-

gery at the University of Missouri at Kansas City School of Dentistry, Consultant in Thoracic Surgery at the Menorah Hospital in Kansas City, and Associate Professor of Surgery at the University of Kansas School of Medicine.

Dr. Herbert C. Miller, Jr., Director of the Children Rehabilitation Unit, Professor of Pediatrics, and Chairman of the Pediatric Department at the University of Kansas Medical Center.

Dr. Robert T. Manning, Associate Professor of Medicine at the University of Kansas.

Dr. William V. McKnelly, Jr., Assistant Professor of Psychiatry and Director of the Psychiatric Consultation Service at the Kansas University Medical Center.

Dr. William J. Cameron, Assistant Professor in the Department of Obstetrics and Gynecology at the University of Kansas.

The program has been approved by the AAGP for 12 Hours of Category I Credit.

Headquarters for the meeting will be the Chaparral Motel.



Dr. Johnson

Monday, July 19

Early Diagnosis

9:00 How to Do a "Check-up"
Robert T. Manning, M.D.

9:25 Early Recognition of Arterial Insufficiency
Jack M. Zimmerman, M.D.

9:50 Prediabetes in Pregnancy
William J. Cameron, M.D.

10:15 Early Recognition of Neurocirculatory Asthenia
William V. McKnelly, Jr., M.D.

10:40 Coffee

11:00 Panel Discussion: Early Diagnosis
Herbert C. Miller, Jr., M.D.,Moderator,
Drs. Cameron, McKnelly, Manning and Zimmerman.

Tuesday, July 20

Vague and Misleading Signs and Symptoms

9:00 The Child With Unexplained Fever
Herbert C. Miller, Jr., M.D.

9:25 Hysteria
William V. McKnelly, Jr., M.D.

9:50 Cardiac Resuscitation
Jack M. Zimmerman, M.D.

10:15 Pelvic Inflammatory Disease and Pelvic Abscess
William J. Cameron, M.D.

10:40 Coffee

11:00 Panel Discussion: Vague and Misleading Signs and Symptoms
Robert T. Manning, M.D., Moderator,
Drs. Cameron, McKnelly, Miller and Zimmerman.

Wednesday, July 21

Psychosomatic Problems

9:00 Hypochondriasis and Depression
William V. McKnelly, Jr., M.D.

9:25 Surgical Manifestations of Psychosomatic Disease
Jack M. Zimmerman, M.D.

9:50 Psychosomatic Stomachaches and Headaches
Herbert C. Miller, Jr., M.D.

10:15 How to Work Up a Patient With Headaches
Robert T. Manning, M.D.

10:40 Coffee

11:00 Panel Discussion: Psychosomatic Problems
William J. Cameron, Moderator,
Drs. McKnelly, Manning, Miller and Zimmerman.

Thursday, July 22

Extremes of Life

9:00 Hormone Therapy in the Menopause
William J. Cameron, M.D.

9:25 Therapeutics for the Aged
Robert T. Manning, M.D.

9:50 Psychiatric Syndromes in the Elderly
William V. McKnelly, Jr., M.D.

10:15 Care and Treatment of Premature Infants
Herbert C. Miller, Jr., M.D.

10:40 Coffee

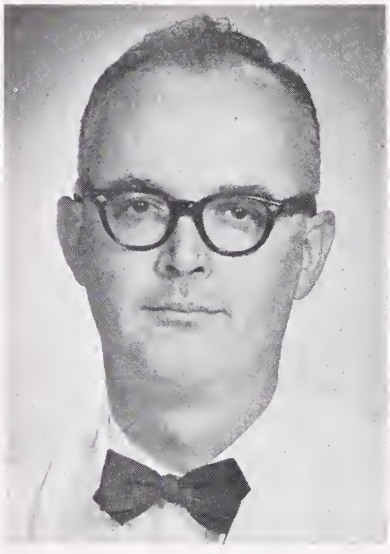
11:00 Panel Discussion: Extremes of Life
Jack M. Zimmerman, M.D., Moderator,
Drs. Cameron, McKnelly, Manning and Miller.



Dr. Zimmerman



Dr. Miller



Dr. Manning



Dr. McKnelly



Dr. Cameron



Dr. Fitz

A Clinical Approach to Aged Patients*

Richard M. Angle, M.D., *Santa Fe*

As modern medical techniques continue to lengthen the life-span, physicians will be called upon to care for increasing numbers of patients who have lived more than 70 years and must be considered "aged". Because of their age, these patients frequently require somewhat different care than do younger adults. The purpose of this paper is to indicate instances in which the elderly patient requires an altered approach to his disease. This approach is based upon some established facts but is largely founded upon the impressions and experiences of years of caring for older patients.

To begin, in treating patients past 70, the physician must make an appraisal. How old is my patient? Certainly chronological age alone is not the sole criterion. We must all know patients in their late 80s or 90s whose alertness and interests place them years younger than some of their contemporaries. And we must all have seen patients who are aged at 65. Many of the former will exhibit normal blood pressure, normal electrocardiograms and chest x-rays and relative freedom from detectable arterial disease. Conversely, many of the latter will show hypertension and evidences of arteriosclerotic heart disease and other extensive arterial disease, suggesting an organic basis for their difference. Certainly the mentally alert individual who manifests interest in his world is less aged than the chronological contemporary with obvious cerebral deterioration. His outlook is young. He needs different care.

A guiding principle in the treatment of the aged is a shift of emphasis. In the infant, child and younger adult, the attempt is always to obtain a cure wherever possible. In the aged patient, beset with multiple diseases, cure of all conditions cannot often be achieved and thus the emphasis becomes one of control. Rarely does the elderly person suffer from a single entity. For example, pneumonia may be secondary to chronic bronchitis or bronchiectasis, and, if severe, may be associated with mild congestive heart failure. Underlying the whole picture may be chronic pyelonephritis. Usually the pneumonia might reasonably be cured, but the other problems at best can only be controlled. To attempt more would be foolish and perhaps even fatal. Likewise in the very aged the surgical approach may have to be modified greatly, still offering comfort to the patient but at greatly reduced risks.

I am reminded of a patient, aged 85, obese and hypertensive, with mild angina pectoris and moderate lymphedema of the legs from previous thrombophlebitis. Always fearful of chronic invalidism and death itself, she developed a palpable mass in the right breast. After a surgical consultation, it was decided to ignore the lesion as long as possible. However, the patient fretted about the mass so much that finally it was removed under local anesthesia and did prove to be a cancer. Since she could no longer feel the lump the patient was no longer frightened and lived comfortably for one and one-half years before succumbing to a cerebral thrombosis. The lesion warranted a radical mastectomy but the patient's needs were

*Presented at the Fourth Annual Neuropsychiatric Seminar, Clovis, New Mexico, October 2, 1964.

best served by a simple excision.

Recently a 93 year old matriarch was admitted to the hospital because of abdominal pain, nausea, fever, and a palpable mass in the right upper quadrant of the abdomen—an obvious case of acute cholecystitis with hydrops of the gallbladder. When there was no reponse to antibiotics after 18 hours, the gallbladder was drained under local anesthesia and the stones were removed. The patient was out of bed in 24 hours and returned to her home in seven days. When seen just a few days ago she was up and active and claimed to feel better than she had felt in years. Cholecystectomy was the treatment of choice, but cholecystotomy was life-saving with little risk.

Providing comfort for the aged patient with the least risk to life should be the goal of treatment. Often this will require the shifting of emphasis from cure to control. Elderly patients seem to react best to the least disturbance in their lives.

A corollary to the "control" approach is the realization that the aged patient is extremely vulnerable to acute disease. In many elderly persons who are living in equilibrium with one or more chronic diseases, a relatively simple event such as pneumonia, gastroenteritis, or influenza may trigger a chain-reaction of complicating diseases which may lead to death in days or a few weeks. I choose to term such catastrophes "cataclysmic deterioration of the aged". The physician must therefore approach the aged patient with caution —on the one hand not to attempt too much—but on the other, not to belatedly do too little.

Drugs In The Aged Patient

Choice and administration of drugs to the elderly becomes extremely important, much as in the very young. Drugs that are well-suited for the younger adult are often too potent or poorly tolerated by older persons.

For example, digitalis may be quite difficult to use in the octogenarian. The average daily dose for an adult, 0.1 gram. of powdered whole leaf, may be toxic to the aged patient and one-half or even one-fourth of this dose may be quite adequate. Years ago a cardiologist in Chicago often referred to the term "digitalis blues". I do not know if digitalis is capable of producing depression but I have seen instances of toxic psychoses in which it most certainly played a part.

Two other classes of drugs should be treated with respect. Narcotics may severly depress an aged patient in doses that would be inadequate in younger patients. This may in part be due to hypoventilation associated with emphysema, so common among the elderly Spanish-Americans, but is probably also in part due to sub-clinical reduction in renal and liver function. Recently I gave a 79 year old female with chronic cor pulmonale who developed acute pulmonary edema an injection containing 25 mgm. of meperidine and 1/200 gr. of atropine. She required support for the next eight hours before the depressant effects were relieved. Caution in the use of narcotics and almost homeopathic doses are often necessary.

Likewise, barbiturates may be capricious in their action on the aged. Only too often they will produce excitement and a transient psychosis instead of the desired sleep. Chloral hydrate remains a safe and effective drug for sedation.

In contrast to the drugs thusfar mentioned, I feel that antibiotics should not be spared in the aged. One must remember that the elderly patient responds poorly to acute infection and may show slight or even no fever and a normal leukocyte count even in the face of overwhelming bacterial infections. One must then pay close attention to the differential blood count as well as other diagnostic parameters and particularly seemingly minor physical abnormalities. Several years ago a 71 year old female developed a cough and pain in the upper right posterior chest. She was afebrile and had no ausculatory findings in the lung but there was slight dullness. However, she appeared to be toxic and was admitted to the hospital where her white blood count was only 4,500 cu. mm., but the differential count showed an increase in the segmented and band cells. A chest x-ray showed extensive opacification of the entire right upper lobe. Only after partial control of the infection with antibiotics did she develop the fever and leukocytosis expected in a severe staphylococcic pneumonia. Delay in starting treatment in this patient might well have been fatal. Maximal dosage of antibiotics should be the rule in treating the aged patient.

Specific Diseases

Age of the patient modifies the therapy of many diseases but there are several conditions commonly seen in which the elderly patient should be

handled differently than his younger counterpart.

I. Obesity

Many patients past 70 are moderately obese. These people have had a lifetime to contemplate their weight and the fact that they have lived 70 plus years without losing excess weight is tacit admission of their unwillingness to reduce. When confronted with such patients I feel that it is unwise for the physician to initiate weight reduction regardless of how medically necessary it might seem. Such attempts are frequently met with resistance, resentment and real anxiety, and may result in a severe depression. In some of my elderly obese patients who are being treated for other diseases which require frequent follow-up visits, I will not even weigh the patient unless forced to by the condition being treated, such as congestive heart failure. I have found that often the patient is more afraid of incurring my displeasure at his failure to reduce than he is concerned about his basic disease. If the obese patient wishes to lose weight I will assist him all I can, but I leave the initiative to the patient.

II. Hypertension

The treatment of hypertension in the elderly is another instance where age greatly modifies therapy. If a patient has reached 70 or over his hypertension is probably mild and not life-endangering. In the younger patient hypertension is treated to prevent the occurrence of the malignant phase or to prevent the severe complications of prolonged high blood pressure. In the aged patient, complications would more likely be due to arterial disease than to the elevated blood pressure itself and it is doubtful that drugs would constitute a protection against cerebral or coronary occlusion. On the contrary it is possible that such complications might follow marked reductions in the blood pressure by reducing the blood flow through diseased arteries. One tends to forget that often the blood pressure rises in response to a physiological need and reducing the pressure may lead to disaster. Where the elderly patient seems to require the reassurance often given by taking a drug, I would favor something relatively mild, such as phenobarbital or at most small doses of reserpine, particularly if tachycardia is present. Diuretics are not indicated in the absence of edema.

III. Acute Myocardial Infarction

Coronary artery occlusion in the aged presents several problems in treatment. Acute thrombosis may be only the final event in longstanding coronary artery disease where satisfactory collateral circulation has developed, and may be tolerated well. However, it may be the final blow to a myocardium already severely compromised by inadequate circulation and lead to rapid death. In any patient, but particularly in the aged, it is vitally important to prevent shock and keep systolic blood pressure above 100 in order to prevent cerebral or other arterial occlusion. The frequent concomitant occurrence of both cardiac and cerebral infarction is certainly not a coincidence. Although age alone does not indicate the use of digitalis in these patients, the elderly coronary patient is more apt to develop congestive heart failure and hence to require this drug.

The decision to use or not use anticoagulants must be made. In general I do not use anticoagulants in the patient past 70 unless he is "young" and develops thromboembolic phenomena. Even then I administer them for as short a period as possible. In the aged the dangers of anticoagulants are greater and the hopes of returning the patient to normal health are certainly less.

IV. Diabetes Mellitus

The treatment of diabetes first appearing in the elderly patient has been greatly simplified by the hypoglycemic drugs. Only rarely must one impose rigid diets and dreaded injections on the patient. Although many may be controlled by dietary restriction and weight reduction, use of the oral drugs may allow greater latitude in diet and lead to a more cooperative patient. At all costs one should prevent hypoglycemia with its risk of cerebral or cardiac damage. It is much safer to allow moderately elevated blood sugar levels so long as acidosis does not threaten than it is to allow hypoglycemia to occur.

V. Strokes

Cerebrovascular insufficiency in the elderly patient is usually due to atherosclerosis of the cerebral arteries, the end result of which is often cerebral thrombosis. Much can be done for the elderly patient who has had a "completed" cerebral thrombosis by relatively simple efforts toward rehabilitation. The institution of a stroke rehabili-

tation program in community hospitals will pay large dividends.

The syndrome of cerebrovascular insufficiency preceeding cerebral infarction has received much attention from vascular surgeons. While undoubtedly of value in selected cases, such surgery is denied many patients because of the lack of facilities in their communities. Anticoagulant therapy for the person threatened by a cerebral thrombosis has been widely used. To date no convincing evidence of the benefit of anticoagulants has been presented, and the dangers of these drugs in the elderly patient are great. Their use seldom seems warranted.

VI. Surgery In The Aged

Age alone should not deny the elderly patient relief obtainable only from surgery. Herniorrhaphy under local anesthesia can give much comfort as can hemorrhoidectomy. The urologists have demonstrated the ability of the aged patient to withstand surgery without undue risk to life. Likewise the gynecologist, by an adroit vaginal hysterectomy or surgical occlusion of the vagina may greatly aid the elderly female with severe cystocele or uterine procidentia. Palliative resection of the colon for malignancy or lysis of adhesions may prolong useful lives. And who can deny the benefits of internal fixation of fractures of the femur or other long bones? As surgical and anesthesiological techniques have improved the risk of many surgical procedures in the aged has become more than acceptable.

Disruption of the Home

Frequently the aged patient reaches a state of deterioration where his family feels that he can no longer live by himself or where the busy offspring can no longer care for him. I should like to make a strong plea that the removal of the elderly patient from his home or accustomed surroundings be delayed as long as possible. The consignment of the aged patient to a nursing home is frequently the first step toward rapid deterioration and death. Depression and dementia often result. In our hospital we have a geriatrics section where the combined ages of 20 patients is astronomical. Those who can think rationally always seem to be verbally wishing for death. The death wish seems to be so strong that I sometimes feel this wing should be termed "the valley of the shadow of death". Of course a time comes when removal of the aged patient to medical surroundings is necessary but such a move should be delayed as long as possible.

Even hospitalization for the treatment of disease should be avoided wherever possible. Less scientific care at home will often produce better results than the finest laboratory-controlled care in the hospital. When hospitalization is necessary, the patient should be allowed up in a chair and walking as soon as feasible and should be allowed to return to his home at the earliest opportunity. Early ambulation and discharge will avoid many cases of depression and dementia.

Finally, the physician must consider what is very likely the most important aspect of the care of the aged—the emotional impact of the physician on the patient. The interest of the physician and his willingness to listen to problems, real or imaginery, a laugh and a compliment will often do more than any drug to comfort and relieve the elderly patient. Above all one should try not to show the irritation that may be caused by the selfish and petty complaints of older people. Just as in show business, the advice, "leave them laughing" applies to the aged. I have found that a house call once or twice a month on the house-confined invalid, plus the judicious use of drugs will produce gratitude and contentment. I warn you, though—this treatment is expensive because it means giving a bit of one's self, but the expenditure is amply repaid by the implicit faith and the smiling face. Try it sometime!

If You Want to Think Better, Feel Better, Work Better, Stand Life Strains Better, Read This . . .

W. Compere Basom, M.D., *El Paso*

To people who are in poor physical condition, this article can probably be of marked benefit.

To you, gentle reader, who considers yourself to be in good physical condition, try this test to see if you are correct. First, however, read this article entirely. Just jog along as if you were out road running like a boxer does to get in good condition (except you can do this right in your own home, just run jogging up and down with the arms and shoulders loose as if you are running) *and time yourself.* If you are in good condition, you can do this for about eight minutes by the clock.

This writer was fortunate in that he attended a luncheon arranged by Dr. Michael O'Heeron at the recent Southwest Regional Meeting of the International College of Surgeons at Houston. Mr. Seymour Lieberman, attorney at law in Houston, spoke about physical fitness.

He is well qualified on the subject, since his article was used as the outline for the article prepared by the President's Physical Fitness Committee of the United States. Mr. Lieberman is 57 years old and looks 47; he is trim and fit. He is able to do the jogging exercise, which is merely running but not traveling, and lecture at the same time without getting out of breath at all. He pointed out that the terrible pressures of today produce a terrific strain on the heart and this situation probably results in the early cardiac deaths that we see today. Therefore, our exercise must condition the body for pressure.

Since Mr. Lieberman was a track athlete he is well acquainted with this kind of exercise. He brought out the fact that in athletes there is a slower cardiac rate than in a person who is not in good condition. The cardiac rate rises less with exercise, more slowly, and returns to normal more quickly.

He pointed out that Socrates when very old was still in very good condition and that over 2,500 years ago a student stated that every morning Socrates did a jog for a while. But although Socrates must have been exercising and keeping fit, it is odd that this was omitted in all commentaries on Socrates philosophies. Despite a thorough search by Mr. Lieberman, he could find no one who had paid any attention to this important fact.

Mr. Lieberman stated that after getting in good condition, his backaches also have disappeared.

He recommends that the way to get into good condition is to do jogging or road work, for this exercises all the major muscles and produces a good physical condition. He found that eight and one-half minutes of this was equal to one mile of running or three miles of walking and therefore one does not need any special equipment at all. One can even do this exercise in the hotel room or almost anywhere. All one must do is 10 minutes a day of this.

A good test would be to see how long you can jog along now. If you can't do eight minutes you are in poor physical condition.

Of course a general thorough physical examination is important with a good cardiac check before one does his exercise, as the exercise is meant for someone in poor condition or someone in good condition to maintain him, but it is not meant for people with special medical problems for whom this would be contraindicated.

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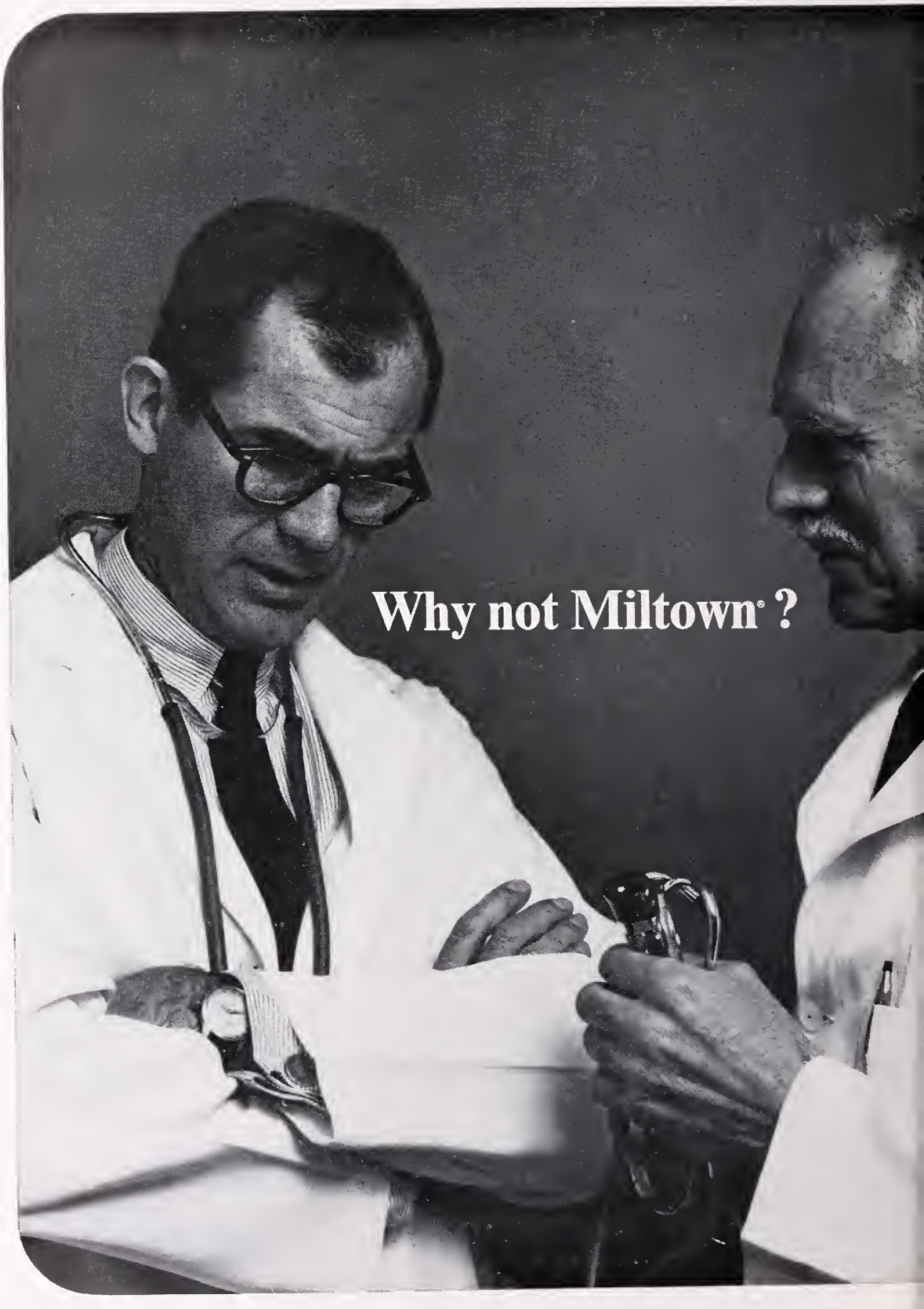
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1965 Scientific Session of the American Cancer Society, Philadelphia, June 16, 1965. Headquarters, Drake Hotel.

19th Annual Rocky Mountain Cancer Conference, Denver, July 16-17, 1965. Headquarters: Brown Palace Hotel.

Eighth Annual Ruidoso Summer Clinic, sponsored by the New Mexico Chapter of American Academy of General Practice, Ruidoso, N. M., July 19-22, 1965. Headquarters: Chaparral Motel.

62nd Annual Meeting of the Western Association of Railway Surgeons, Alameda Hotel, Avenida Juarez 50, Mexico City, October 27-30, 1965.

15th Annual Meeting of the Southwest Obstetrical and Gynecological Society, Arizona Inn, Tucson, Oct. 28-30, 1965.

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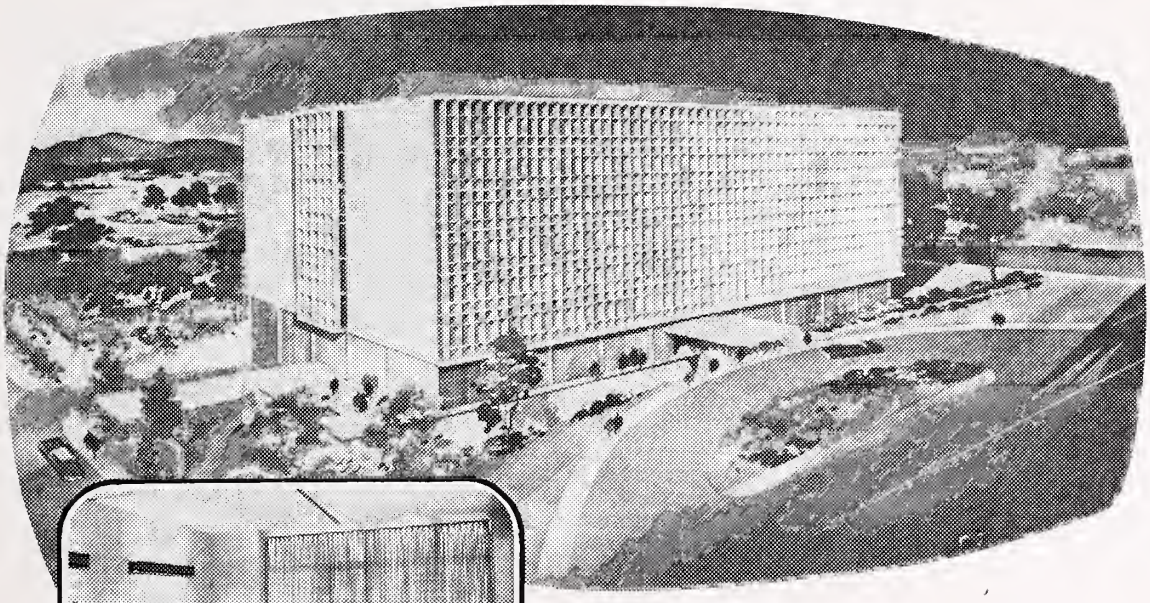
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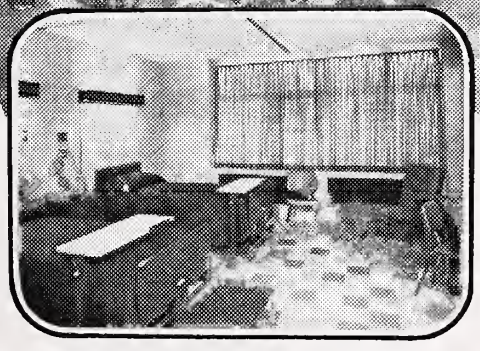
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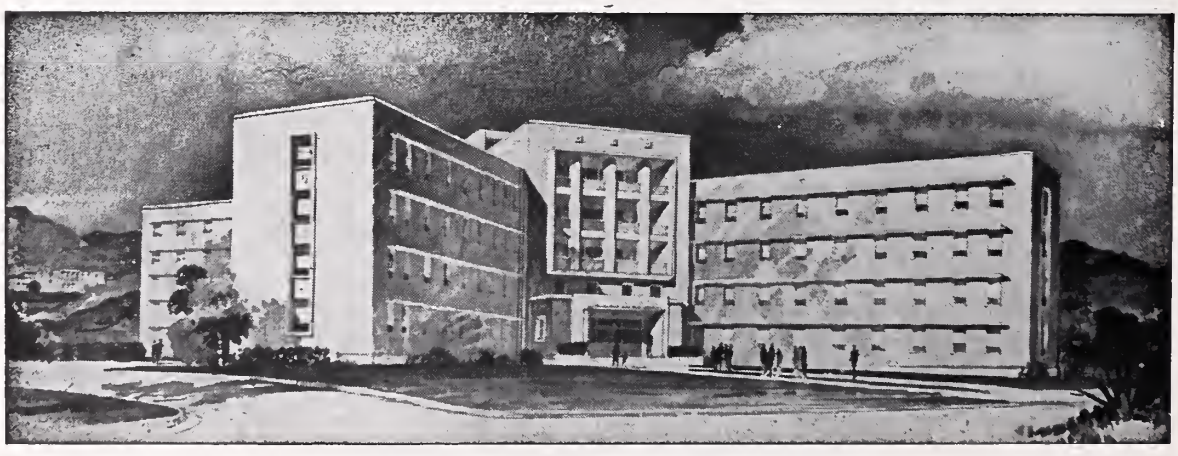
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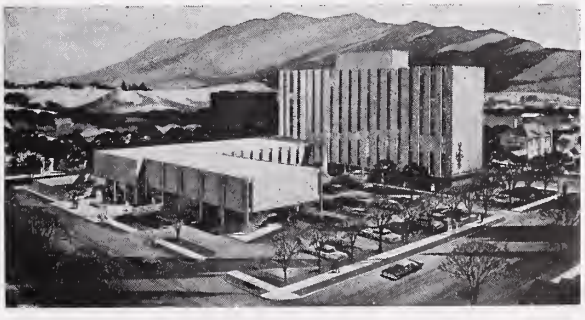
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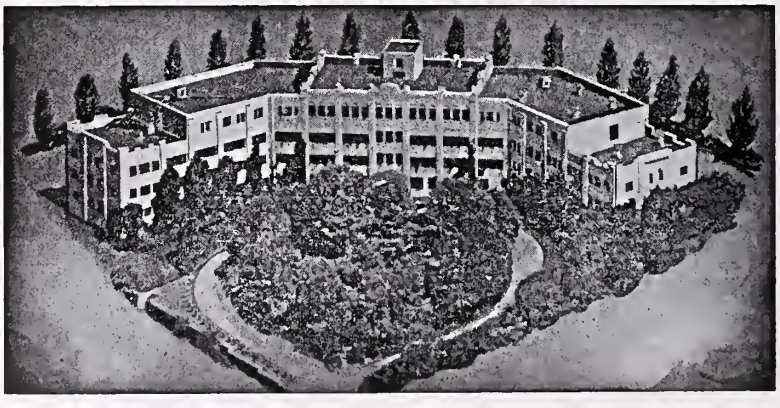
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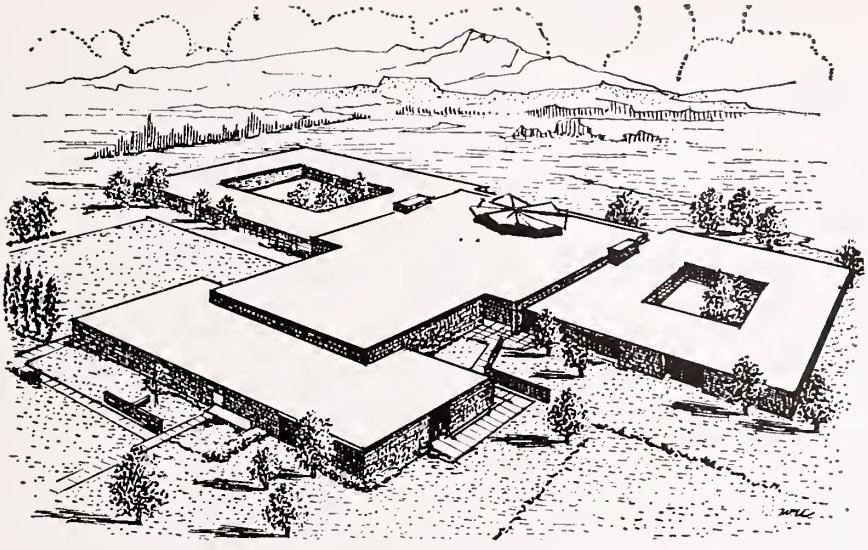
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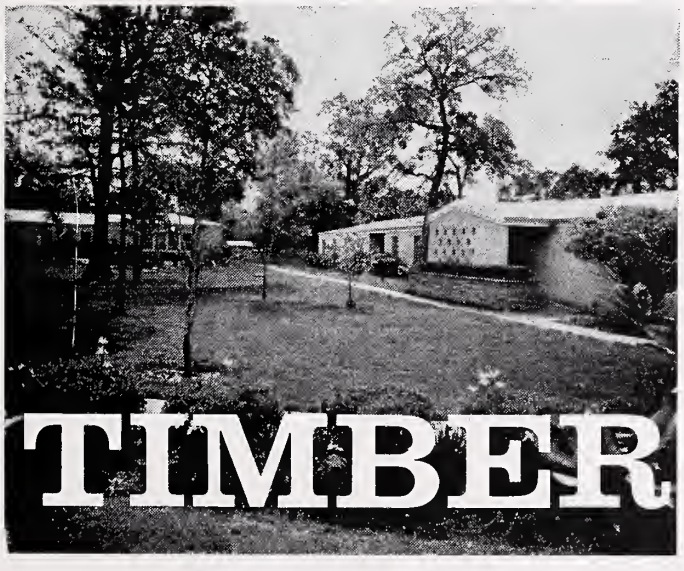


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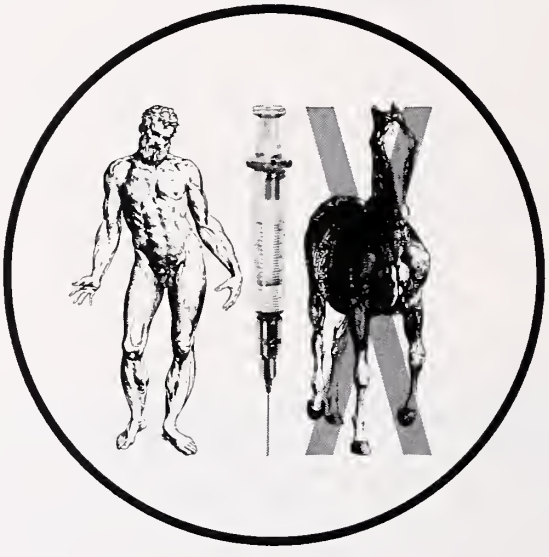
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References: 1. Rubbo, S. D., and Suri, J. C.: Brit. M.J. *2*:79 (July 14) 1963. 2. Rubinstein, H. M.: Am. J. Hyg. *76*:276, 1962. 3. McComb, J. A.: New England J. Med. *270*:175 (Jan. 23) 1964. 4. Effective tetanus protective level established by Sir David Bruce. 5. Editorial: Tetanus Immunization, JAMA *161*:883, 1956.

The Fate of the Elderly Patient in a Psychiatric Hospital*

Leonardo Garcia-Bunuel, M.D.,** and George Tippin, ACSW***, *Pueblo, Colorado*

The fate of the institutionalized aged is largely dependent on the ability of institutions to stop or even reverse the process of social rejection which has taken place in the community and has finally culminated in hospitalization.

Well over half the institutionalized aged in this country are housed and presumably cared for in state mental hospitals. State hospitals are not, by and large, dynamic organisms. Too often, the public and our legislative bodies see them as institutions providing inexpensive warehousing for human refuse. Budgetary appropriations tend to reflect this attitude so that, of necessity, state institutions have become very skillful at classifying the human beings forcibly entrusted to their care into categories which will facilitate proper storage at minimum cost. Thus, we all have become painfully familiar with terms such as "acute", "untidy", "disturbed", "chronic", "regressed", etc. As far as we have been able to ascertain, the term "geriatric" is a very poorly defined one outside of institutional jargon, where it has come to mean anyone more than 65 years of age. There is some evidence to indicate that in states where the age of retirement has been fixed for civil servants at 70, any institutionalized person below 70 is not considered to be "geriatric." Ages of compulsory retirement are fixed at points varying between 60 and 75, all being equally arbitrary and unscientific. C. Northcote Parkinson has stated that, "Where the retirement age is fixed at 65, the defenders of this system will always have found, by experience, that the mental power and energy show signs of flagging at the age of 62. This would be a most useful conclusion to have reached had not a different phenomenon been observed in organizations where the age of retirement has been fixed at 60. There, we are told, people are found to lose their grip, in some degree, at age 57 . . . It would seem, in short, that efficiency declines at age of R (retirement) minus three, irrespective of the age at which 'R' has been fixed." We are confronted with a bitter paradox: at a time in our history when people are living longer and remaining physically and mentally healthier further and further into advanced age, the age of compulsory retirement is decreasing rapidly. As our society moves forward, younger and younger rejects flock into our institutions. Our geriatric population is getting younger, a cheerful sounding statement were it not tragic because of its long-range consequences.

Having found that one is useless and that one has been officially declared so by commitment to an institution is not an easy situation to accept nor a charming fate to adjust to. The will to live is, indeed, shattered. One needs not wonder as to the reason why so many so-called geriatric patients cease to live within a couple of weeks of admission to an institution. You are all familiar with the fact that these early deaths cannot be, in most cases, adequately explained by postmortem findings. To be sure, the body machinery was somewhat worn out but none of its parts show evidence of having

*Presented at the Fourth Annual New Mexico Neuropsychiatric Seminar, Clovis, New Mexico, October 2, 1964.

**Director, Clinical Program, Colorado State Hospital, Pueblo, Colorado.

***Colorado State Hospital, Pueblo, Colorado.

broken down. But, if one has nowhere to go, why keep on moving?

The shock of sudden and drastic rejection and isolation results in severe depression. The need to adjust to a radically different way of life places enormous demands on the organism when its vital tools are beginning to wear out. As people become useless to the society in which they live, they are denied the opportunity of contributing to their fellow beings. The man who is denied the opportunity of making decisions of importance begins to regard as important the decisions he is allowed to make. Thus, our young geriatrics become fussy about minor details and may endlessly hesitate before taking any step. Their nuisance value increases in direct proportion to our perception of them as useless burdens. Thus, all too often, they are seen as hostile, unattractive, clumsy, grouchy, resistive to change, obstreperous and, eventually helpless.

When this point is reached, warehousing—more kindly known as hospitalization—becomes mandatory. This has many advantages. Two of them are very obvious: hospitalization eliminates the elderly from their immediate environment and, simultaneously, provides a good rationalization for the elimination process, thus allaying guilt. The state institutions assist in this process by immediately attaching a label to the new guests, carefully chosen from the latest edition of the APA's Diagnostic Manual. It must be noted that such manual, under Code Number Y00-000 (disease none) provides an opportunity to every institutional (or should we say "institutionalized"?) physician not to become a part of the conspiracy that has declared old age to be a mental illness. This opportunity is seldom, if ever, taken advantage of by our "institutionalized" physicians.

Storage having been thus accomplished, we soon find our elderly patient housed in a more or less attractive building consisting of an accumulation of wards provided with a number of dormitories all converging upon a rather wide expanse of uninspiring space called the "day hall." Here, our patient will spend long hours of forced wakefulness with many others who, like him, are in storage and are likely to so remain until delivered by death. This day hall is usually flanked by a glass-enclosed nurses' station from which overtaxed nursing personnel can simultaneously watch the patients and frantically go about their many bureaucratic chores, one of which is the writing of "progress notes." A common notation is, "Evidence of paranoid ideation; complains of being continuously watched." Thus, what was not an illness is now becoming one, for the elderly one is being made to fit our pre-conceptions about him.

The elderly are not placed in these wards on the basis of a communion of interests or similar social background. Neither their past nor their future, as they themselves perceive both, are used as criteria for housing them together. Rather, they are grouped in terms of the problems they present to their caretakers. Thus, we have our "physiological" classifications where all post-stroke patients are in Wards 1 and 2—(sexes, let us not ever forget it, must be kept separated at all costs)—all those with bed sores will be housed in Wards 3 through 6, all diabetics share quarters in Wards 7 and 8, etc. The whole person fades away; one symptom or complex of symptoms receives concentrated attention. While our patient used to associate with people who enjoyed hunting, folk-singing and criticizing Barry Goldwater, he is now forced to live with people who are vegetarians, love the Beatles and are members of the John Birch Society. The only common bond now is the daily donation of a clean urine sample and the anxious wait for news of how many "pluses" of sugar were determined to rate their urine in a new and bewildering performance scale. Yet, we wonder why our patient has become so concerned with physical symptoms.

While the elderly person does hide behind a shell of impassive gruffness which pushes away many attempts at treatment, such a shell is of our making. The elderly person in our society is rejected as a useless person no longer capable of producing any useful item. The elderly are quick to pick up these feelings and reflect them back to those who do attempt to offer services.

All too often treatment programs for the elderly are developed with little consideration of their needs or wants. Many times hobbies are over-rated as a treatment technique, while in fact, hobbies are the product of the young to the middle-aged. How many elderly persons do you know who took up a hobby upon reaching old age? The normal older person gives up the hobby. The reason for this is simple. The elderly feel useless and the pro-

duction of useless items only reinforces their feelings of uselessness. The elderly have but little regard for the production of things that look nice but serve no worthwhile purpose. At the same time, the elderly person may well surround himself with all manner of useless items; yet to discard these items as useless would be the same as discarding himself.

If the elderly patient in a psychiatric hospital is thought of as hopeless, the entire treatment program takes on the aspects of terminal hospitalization. Treatment is geared towards maintaining the patient at the current level of functioning. The elderly, however, are difficult to maintain and patients deteriorate. Such was the fate of the elderly at the Colorado State Hospital prior to 1962. In March 1962, the hospital underwent geographic decentralization and the philosophy of the hospital shifted from that of terminal hospitalization for the elderly to active treatment. Treatment is now directed towards the restoration of physical and mental health in the elderly with return to community living as the goal of treatment.

Treatment programs for the elderly have developed that are not of a traditional nature but directed towards the treatment of the whole patient. The patients are so involved in activities and treatment programs that they do not have either the time or the need to regress or deteriorate. Chair and bedridden patients are ambulated. Care is taken not to fall into the trap of meeting all of the patient's needs. When all of a person's needs are met, he loses respect for himself, becomes more demanding and, at some point, is then rejected by the staff.

In one of the geographic divisions of our hospital the occupational therapy program for the elderly patients has taken the form of making only those things that can be used in the patients' living area, or in the occupational therapy shop. Several elderly women spent months making a rug. Some cut the strips of material, some sewed them together, others crocheted the rug. The product was useful as it was placed on the floor of their day room; they worked together and benefited from the socialization of the project. The men, on the other hand, made a coffee table which is the center of one of the living areas. Perhaps the most outstanding program was the summer project of the men preparing, planting and maintaining a garden while the women made aprons, pot holders and other things to be used in cooking. The elderly women finished by cooking the produce from the men's garden, baking bread and then both groups had a party to enjoy the products of each other's labors. The importance of role functioning need only be mentioned.

In the same area, these elderly patients were also involved in a type of group therapy. They were not forced to forget the past and talk only about the present but were allowed to start where they were and move to the present. Encouraging a patient to recount his experiences traveling from Missouri to Colorado in a covered wagon made him feel important; yet, by relating these experiences to those of others in the group and to present modes of travel prevented the patients from unnecessarily dwelling in the past and brought the group in touch with the present. Having such a large past and such a small future, the present is invested with profound meaning.

In planning for community living, discussions were started with patients' remembrances, relating them to their current circumstances and ending with visits to the community and to the facilities for care within the community. It is of interest to note that the general store of 50 years ago is not markedly different from the large modern supermarket of today except that today's supermarket is larger and usually does not have coal oil in a gallon can with a potato stuck on the spout. On the other hand, the nursing care facilities within the community are now a far cry from the old County Poor Farm which the elderly remember.

The same may be said for activities. While card games and puzzles are a little work for the employees, they are of much more value when the employee takes the time to help with the puzzle or to play with the patients the card games they remember. Also there is no reason why the wheelchair patients need only to watch others square dance when the employees can push the wheelchair patient in the square dance. A large ball, a group of elderly patients and an enthusiastic employee can produce deafening laughter. A bouncy polka, an enthusiastic employee, and the 70-year-old man will end up dancing with the 80-year-old woman, usually after he has first danced with the young female employee. A State

Fair or carnival is enjoyed by the elderly just as much as by the young, and there is no reason why the wheelchair patients should be left at home. Food is always better at a picnic and the 77-year-old man may be a pretty good chef. The known feeding problem may, and did, consume two hamburgers with all the trimmings plus baked beans, potato chips and an ice cream cone. This was without stomach upset the next day or any other time, for that matter.

It is thought by some that the elderly want to be free from the din of young children. This is not true. At a party to which the children of the employees were invited, an elderly man usually out of contact and delusional, related very well and quite rationally to a four-year-old girl, who, by the way, ate the most of his cookies. Our Children's Treatment Center is being constructed close enough to the Geriatrics building to allow the elderly patient to sneak a swing when no one is looking. Our state institutions should not separate those who are beginning to face life's challenges from those who have already faced them.

It is thought that the sexes must be kept separate in a psychiatric hospital. By placing the elderly men and women where they can intermingle, the old man will shave and be a little more careful of his zipper, while the little old lady will wear her teeth and comb her hair. Both will improve in their table manners and language. It is not unknown that two octogenarians will hold hands and there may even be a romance or two.

The fate of the elderly patient in a psychiatric hospital is dependent on the imagination, ingenuity and inventiveness of those charged with their care and treatment. Equally important is the necessity for those giving treatment to have the clinical freedom and encouragement to use other than traditional treatment methods as well as the traditional medical care needed to restore the elderly patient to his highest level of physical functioning.

Treatment must be goal directed, starting where the patient is. The whole patient must be treated with a variety of treatment techniques. The elderly post-stroke patient will be using both hands while kneading bread or hoeing a garden. The elderly women who are jointly making a rug for their living room are working together, socializing and, at the same time, are doing something useful. After the bedridden patient is helped into a wheelchair there must be some place to go.

If the elderly patients are involved in an active and comprehensive treatment program there is neither the time nor the need for them to regress or deteriorate. Physical illness is treated sooner, but more importantly, illnesses are prevented as the bed patient is turned and the untidy is panned often enough to prevent bed sores. The patient is kept in contact with reality through discussion, visits to the community, and contact with not only each other but with younger persons.

But, let us go back to the traditional "physiological" classification, a form of management likely to prolong biological life. One day, however, death will be lurking around. Our elderly patient will, at this point, be removed to an area full of chromey gadgetry where nurses shuffle along in soft-soled shoes and the caretakers talk in whispers to each other. This area is likely to be located close to the hospital's morgue and has, otherwise, easy access to the outside. Dignified hearses are seen, lying in wait, not far away.

Once more, in the long journey to the grave, the elderly has been rejected. And this, when the need for friends, for known faces, evoking aromas, familiar voices, is greatest. He has been removed from that ward in which, through an enormous effort, he had become a member of a new family. Now, he is confronted with a smooth running, impersonal machinery which will possibly add, through the use of sleek looking gadgetry, a few minutes or hours to a life, which if he is to understand the message, has been a burden to all but himself. He will soon be buried with pomp and circumstance commensurate with someone else's interest to pay for the disposal of his remains. The funeral director will do a fairly impressive job, regardless of how much money is available to pay for the performance. This final ritual having been taken care of, relatives, friends and the hospital administration will feel their guilt relieved to a considerable degree. Our patient, on his way to heaven, will wonder why his departure to a better life was not accomplished with more dispatch and less fuss.

The disposal of the elderly is related to the need

of making room for the younger. As more and more youngsters enter the labor market, increasing numbers of grownups are being forced into idleness. Child labor laws have been, in recent times, bitterly attacked. It is assumed that such laws force many youngsters into idleness which results, among other ills, in increasing juvenile delinquency rates. This may well be so. Yet, the sooner a youngster is allowed to enter into the labor market, the sooner the so-called elderly will become refugees, displaced persons, or call it what you will. And their wisdom will go to waste. And they will be laid away into human warehouses. A few years ago, 75 years was time when one was declared to be "geriatric." Today, most states have established 65 years of age as the boundary between reasonable productivity and positive uselessness. In another 20 years we shall be old at 55. A serious problem is then confronting us: what are we ourselves to do when nearing the retirement age we have fixed for others? To quote Parkinson once more, "It will be obvious at once that our own case is entirely different from any other case we have so far considered. We do not claim to be outstanding in any way but it just happens there is no possible successor in sight. It is with genuine reluctance that we agree to postpone our retirement for a few years, purely in the public interest . . . We knew how to make our predecessors retire. When it comes to forcing our own retirement, our successors must find a method of their own."

AAGP President To Speak At Ruidoso Meeting

Dr. Amos N. Johnson, Garland, North Carolina, President of the American Academy of General Practice, will speak at a banquet on the night of July 21 at the Eighth Annual Ruidoso Summer Clinic, sponsored by the New Mexico Chapter of the AAGP in Ruidoso, New Mexico, July 19 to 22, 1965.

Also speaking at the banquet will be Dr. Reginald Fitz, Dean of the University of New Mexico School of Medicine.

Faculty members at the popular summer meeting, high in the pines of the Sacramento Mountains, are being provided by the Post-Graduate Medical Education Department of the University of Kansas Medical Center. Dr. Jesse D. Rising is Director of the Department.

The meeting is approved for 12 hours of Category I Credit. The Registration fee will be $25 but it will be waived for Interns, Residents, Armed Service and Public Health Service Personnel, Faculty Members and Medical Students. Headquarters will be at the Chaparral Motel.

Subjects for the four-day meeting will be Early Diagnosis, Vague and Misleading Signs and Symptoms, Psychosomatic Problems, and Extremes of Life.

Acute Fatal Bronchitis and Pneumonia Associated With Exposure to Freon*

Alan R. Laurain, M.D.,** and Robert Schoenfeld, PhD., *** *Albuquerque*

Introduction

Pure Freon gas is virtually non-toxic, but, when exposed to temperatures above 233°F., is converted to hydrogen chloride, hydrogen fluoride, chlorine, and phosgene.[1] Such conversion products have resulted in illness of healthy young male smokers, flame photometer operators, and others exposed to Freon and open flame in the closed atmosphere of nuclear submarines.[2] Future space pilots within closed atmosphere systems may also be exposed to this hazard. This is a report of exposure to Freon-12 (Dichloro-Difluoro-Methane) of a 68-year old male with Parkinson's disease and pulmonary emphysema. The gas was liberated from a refrigerator when he punctured the freezing coil with an ice pick.

Case Report

This 68-year old, unemployed, white male, with known, slowly progressive, Parkinson's disease since 1950, was admitted to the Veterans Administration Hospital, Albuquerque, New Mexico, on July 9, 1959. A laceration of the right forehead, sustained during a fall to the sidewalk shortly before admission, was present. He had experienced shortness of breath for several years, attributed on prior hospitalizations to pulmonary emphysema, but had never had paroxysmal nocturnal dyspnea or orthopnea. He lived alone in a one-room apartment near the residence of a daughter.

He had been treated for bronchitis a number of times, most recently in November, 1958. During these admissions multiple sputum studies and gastric washings, done because of a history of arrested pulmonary tuberculosis (1923-1933), failed to reveal tubercle bacilli on culture. He was hospitalized from November 5, 1957 to February 26, 1958 for treatment of a right malar eschar, presumably the result of a fall on a hot stove. Chest X-rays taken during this and prior admissions showed no changes suggestive of inflammation.

The laceration prompting the current admission was sutured and healed promptly. A chest film taken the day after admission showed calcification in the left midlung field and features suggestive of pulmonary emphysema, both of which had been noted on many previous films. An ECG was interpreted as left heart strain and/or Digitalis effect. Laboratory studies revealed: WBC 7,100 with 54 per cent neutrophils, 42 per cent lymphocytes, and four per cent eosinophils; hematocrit 46.5 per cent; hemoglobin 14.9 gm. per cent; urine 1.027 with two to four white blood cells per high power field, no albumin or casts. He was discharged at 3 p.m., on July 15, 1959 to return home.

The patient was readmitted to the hospital at 4:30 p.m., July 16, 1959, unaccompanied, confused, and completely unable to give a history. Previously he had been able to communicate moderately well. The rectal temperature was 101.6° with a pulse of 116 per minute. First degree burns were noted on both forearms and over the left thoraco-abdominal region. Six hours after admission, the temperature rose to 103° F., and the pulse was 138. The abdomen was soft and the

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chest clear to auscultation. Local warm soaks were applied to the right forearm. The temperature dropped to 101°F. following sponging and aspirin. Readmission laboratory studies revealed: WBC 18,200 with 74 per cent neutrophils, and 26 per cent lymphocytes; hemoglobin 21 gm. per cent; hematrocrit 62 per cent; Sodium 132 mEq/1, Potassium 4.2 mEq/1, CO_2 15 mEq/1, Chlorides 109 mEq/1, BUN 66 mgs. per cent; urine albumin three+ with six to eight hyaline casts, three to five granular casts, and one to two epithelial casts per high power field.

Profound shock developed on the morning of July 17, 1959. The pulse and respiratory rates were markedly elevated. An IV cutdown was made and he appeared to respond to fluids, metaraminol, and hydrocortisone. Moist rales were heard in the lung bases bilaterally, and large doses of penicillin and streptomycin were started. A portable chest film showed an elevated right diaphragm, but no frank evidence of pneumonia. Subsequent films were unsatisfactory because of motion, but revealed no gross evidence of consolidation. The temperature was 104°F., rectally.

On July 18, 1959, spiking temperatures continued, and shock became much worse. He was digitalized even though the ECG had not changed from one taken earlier, except for sinus tachycardia. Levophed was added to the intravenous fluids, but wild variations in blood pressures continued. That morning a daughter volunteered the information that the patient had been attempting to defrost his refrigerator with an ice pick on the night before readmission.

Coarse rhonchi were heard by his physicians and nurses on July 19, 1959. Suctioning of the trachea by the nurses initially produced only a small amount of rusty sputum, but later progressively larger amounts of yellow purulent material were removed. Blood chemistry revealed: Sodium 128 mEq/1, Potassium 5.8 mEq/1, CO_2 12 mEq/1, Chlorides 93 mEq/1. The urine output became progressively lower daily, and on the day of death no urine was produced. The BUN was 110 mgs. per cent. Cheyne-Stokes respiration developed and he died at 1:00 p.m., on July 19, 1959.

Investigation of Decedent's Apartment

On July 21, 1959, the senior author, accompanied by a hospital engineer and legal authorities, found a three mm. puncture in the freezing coil of his four cubic foot refrigerator. The point of an ice pick on an adjacent sink counter fit perfectly into the hole. Immediately opposite the refrigerator was a 30 gallon gas water heater which came on once during the investigation. The diagonal distance between the burner and the hole in the coil was between four to five feet.

Testimony of relatives and neighbors revealed: that the decedent always kept the doors and windows closed; he was last seen at 10:00 p.m., July 15, 1959 by a daughter who noted that the refrigerator was working perfectly; he was heard walking around in the kitchen, separated from the next apartment by a very thin partition, until about midnight; he was found the next day at 11:00 a.m., by a daughter, lying across the bed unable to state what had happened.

Autopsy Findings

Burns involving most of the ventral surfaces of both forearms were noted. A similar burn along the left lower rib margin measured 10 x 6 cm. In a number of places there were hard, reddish-brown eschars. The total burned area was much less than one per cent by the rule of nines. Careful inspection of the face failed to reveal burns. Bilateral cut down wounds at the malleoli, one containing a plastic catheter, were present. Pertinent gross pathological changes were confined to the lungs and brain. The right lung weighed 600 and the left 550 gms. The trachea and both major bronchi contained abundant extremely bright yellow viscid exudate which could be readily removed, revealing diffusely reddened shiny mucosa. In the dependent portions of the lungs bilaterally, the exudate completely occluded the smaller bronchi. On section these areas were dark red and sank slowly in the fixative. The anterior aspect of the lungs was pink and crepitant, with scattered anthracotic areas and a moderate amount of frothy, somewhat blood-tinged fluid exuding from the cut surfaces. One of the left hilar lymph nodes was enlarged and markedly calcified. A calcified nodule about eight mm. in diameter was present in the upper portion of the left lower lobe. The brain weighed 1500 gms. and grossly showed evidence of slight edema. The major cerebral vessels were thin and delicate.

Microscopic sections through the cerebral basal ganglia revealed focal encephalomalacia and vas-

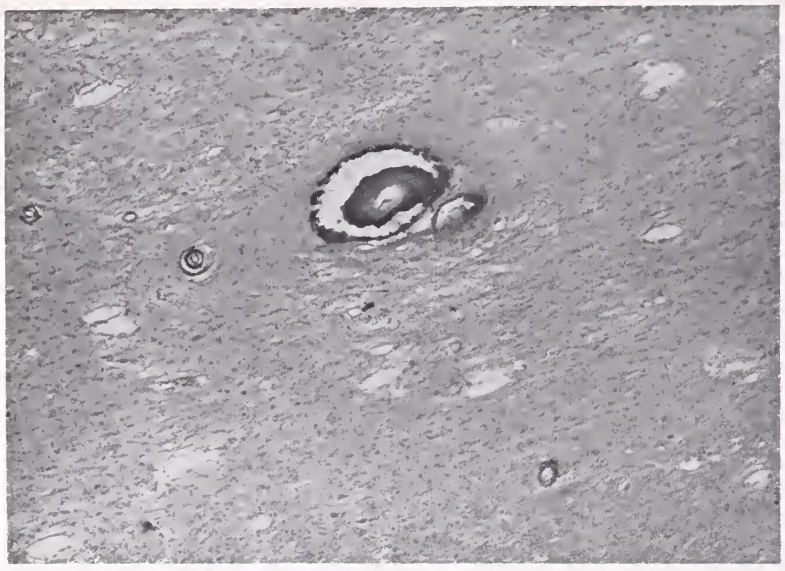


Fig. 1 Basal ganglia H & E x 16 showing arteriolosclerosis and encephalomalacia.

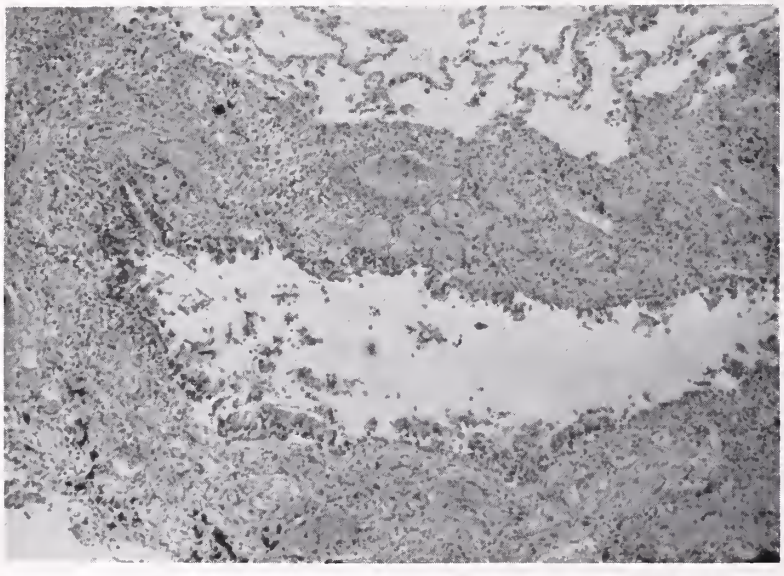


Fig. 2 Small anterior bronchus, right middle lobe H & E x 18 with marked vascular engorgement and absence of inflammation in the surrounding lung parenchyma.

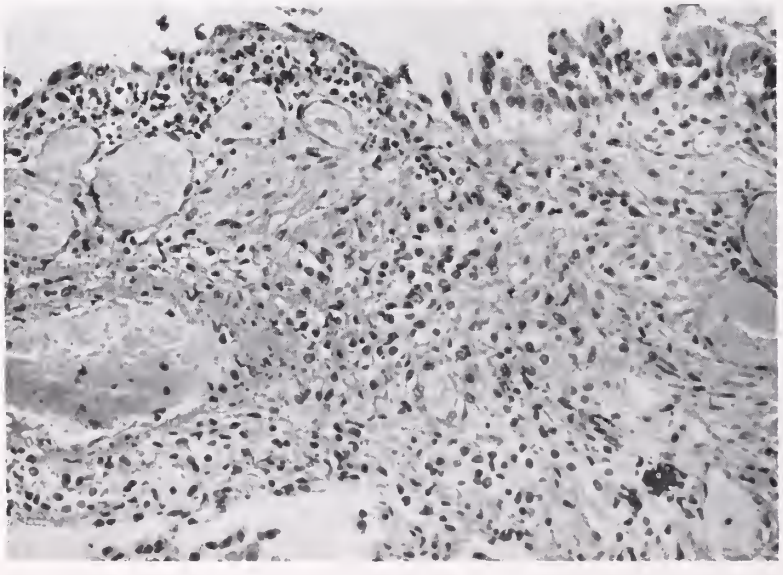


Fig. 3 Same bronchus as Fig. 2 H & E x 100 showing early ulceration at left and desquamating cells at right.

cular changes regarded as typical of Parkinson's disease (Fig. 1). Most sections through the lungs revealed extensive confluent bronchopneumonia involving the dependent portions. Sections through the anterior aspect of the lungs revealed many small bronchi showing marked submucosal vascular engorgement, interstitial neutrophil infiltration, and focal ulceration of the overlying mucosa (Figs. 2, 3, 4). The surrounding lung parenchyma was strikingly free of inflammatory reaction.

Moderate pulmonary emphysema, focal collections of "heart failure" cells, and areas of edema without cellular infiltration were noted also, predominantly in the anterior portions of the lungs.

Discussion

The findings at autopsy and the chain of circumstances eventuating in death from bronchopneumonia strongly support our belief that toxic degradation products of Freon-12 were inhaled by the decedent. Freon-12 has been known to decompose within appliances, but internal corrosion soon causes a cessation of function.[3] Such an event is quite unlikely in this case, but could not be excluded because the refrigerator was disposed of before it could be examined.

Freezing of bronchial mucosa from inhalation of cold air and Freon-12 is quite unlikely in the absence of "burns" around the mouth. Exposure to pure Freon-12, in a concentration of 20 per cent by volume, is quickly manifested by generalized tremors in animals, including primates.[4] Thus, inhalation of Freon-12 could very well have aggravated the already severe motor impairment from Parkinson's disease. One might argue that the skin burns alone were at fault, but, by a very generous estimate, only 240 square centimeters were involved, which cannot explain the presence of severe, progressive, irreversible shock of the type frequently seen after exposure to noxious gases.[5] This case has some features in common with fatal chronic bronchitis,[6] but many differences permit exclusion of that diagnosis. Furthermore, the patient presents a past history of excellent resistance to respiratory infection. In 1958 he was admitted with a history of lying semicomatose in his apartment for three days before being hospitalized, and still not developing pneumonia. Since that time he had been free of respiratory symptoms until the terminal admission.

Before Freon-12 was used on a large scale, numerous ingenious experiments were performed to mimic conditions likely to be encountered with slow or rapid leaks from commercial and household refrigerators in the presence or absence of flames and hot surfaces.[7,8] One of these[8] indicates that even a pilot light can produce sufficient degradation of Freon-12 to result in symptoms in guinea pigs within seven minutes and death after exposure of four hours. With an ordinary range burner and uniformly distributed Freon-12 in an unventilated room, two guinea pigs developed symptoms in one minute and died in nine and

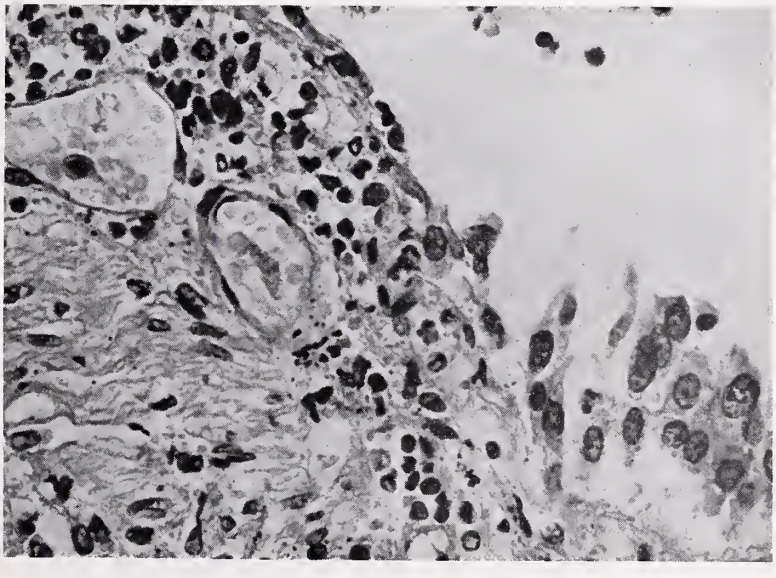


Fig. 4 Detail of area shown in Fig. 3 H & E x 160.

one-half and 12 and one-half minutes respectively.[8]

The only ventilation in the patient's apartment was through the flue of the gas water heater. Undoubtedly he turned toward his right after puncturing the coil, thus exposing his left thoracoabdominal region to the escaping gas. This would have brought his face close to the water heater. Perhaps he fell in the kitchen because of Parkinson's disease, and lay there for a while before proceeding to the bed where he was found.

Freon-12 is much heavier than air and would flow quickly downward to the level of the burner. It interferes with combustion[7] and the top of a water heater burner is above the critical conversion temperature for at least one minute after cutoff.[9] The degradation products are also much heavier than air. Therefore, if Freon-12 reached the burner shortly before it went out, the possibility of substantial quantities of noxious gases entering the room appears quite good.

Theoretical calculations listed in Table I are based upon conversion percentages from a standard text[1] and assuming: (a) Uniform dispersion of 20 ounces or originally liquid Freon-12 throughout the 6 x 6 x 8 foot kitchen; (b) no loss through the flue; (c) and loss of all but 1/20 of Freon-12 and its degradation products through the flue.

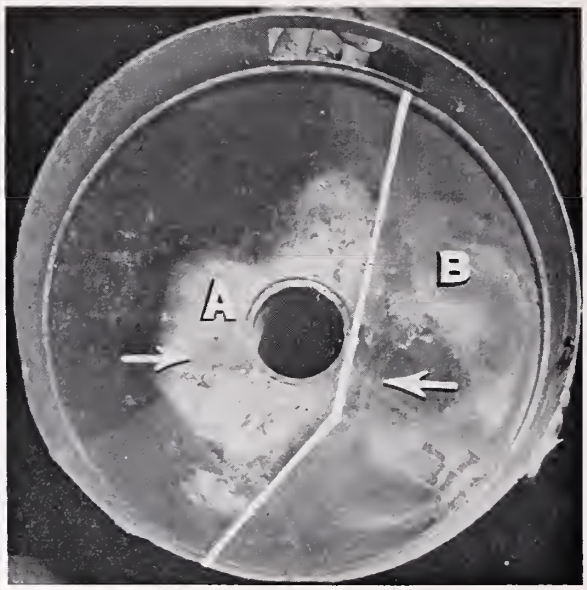


Fig. 5 Dome of water heater combustion chamber showing corrosion centered around the flue opening. The arrows indicate sites of sampling for chemical analysis. White corrosive deposits with dark areas of rust are seen at "A". Lime deposits and rust with a flow pattern resulting from a leak through the welded joint between the flue and the top of the combustion chamber are indicated at "B". The chamber access opening shows at the top.

TABLE I

Toxicity Levels and Calculated Room Concentrations of Freon Conversion Products
All Values in Grams Per Cubic Meter

	Toxicity	Maximum	1/20th of Maximum
CCl_2F_2	—	—	—
HCl	2.8-5.5	18.4	0.90
HF	0.15-0.40	8.6	0.43
$COCl_2$	0.2-	1.4	0.07
Cl_2	2.8-5.5	0.29	0.01

About one year after the accident the water heater in the decedent's apartment developed a leak similar to those which are known to result from corrosion (Fig. 5) by hydrofluoric and hydrochloric acid condensates occurring with degradation of Freon-12.[10] Chemical analyses of the gas heater parts for water soluble fluorine[11] revealed the following percentages by weight: Burner Canopy 5.14, top of water heater flue 1.54, combusion chamber "A" 3.09, combustion chamber "B" 1.32, fluepipe within tank 0.03. These fluorine levels can only be explained by conversion of Freon-12 into its degradation products. Of note, in this regard, is the very low level within the tank proper and the lower level, apparently from leaching, at "B" in Fig. 5. The metal and natural gas in this area can be excluded as sources of fluorine.[9,10] The possibility of roach powder as a source of fluorine was excluded by analysis of corrosion from "A" (Fig. 5) for sodium and potassium. Fluorine analyses by steam distillation of lung, liver and kidney revealed only trace amounts in the current case and in a control stored in 10 per cent neutral formalin for a similar period.

Summary: A case of acute fatal bronchitis and pneumonia associated with exposure to Freon-12 and, in all probability to its degradation products, is reported and discussed.

Acknowledgements:

We are indebted to Miss Sabhia Thannoun, Public Works Laboratory, Albuquerque, New Mexico, for some of the fluorine analyses. We wish to thank the many individuals, too numerous to list, who generously gave of their time and knowledge, during the course of this investigation.

Illustrations by: Howard Toenniges.

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Coming Meetings

19th Annual Rocky Mountain Cancer Conference, Denver, July 16-17, 1965. Headquarters: Brown Palace Hotel.

Eighth Annual Ruidoso Summer Clinic, sponsored by the New Mexico Chapter of American Academy of General Practice, Ruidoso, N. M., July 19-22, 1965. Headquarters: Chaparral Motel.

International Orthopedic Seminar, sponsored by the American Physicians Fellowship, to be held in Israel Aug. 4-15, 1965. Please direct all inquiries and reservations to the American Physicians Fellowship, 1622 Beacon St., Brookline, Mass. 02146.

New Mexico-El Paso Chapter of the American College of Surgeons, Ruidoso, N. M., September 11-12, 1965. Headquarters: Chaparral Motel.

62nd Annual Meeting of the Western Association of Railway Surgeons, Alameda Hotel, Avenida Juarez 50, Mexico City, October 27-30, 1965.

15th Annual Meeting of the Southwest Obstetrical and Gynecological Society, Arizona Inn, Tucson, Oct. 28-30, 1965.

47th Annual Meeting, Southwestern Medical Association, Sheraton Motor Inn, El Paso, November 4-6, 1965.

99th Annual Session of the Texas Medical Association, Austin, April 14-17, 1966.

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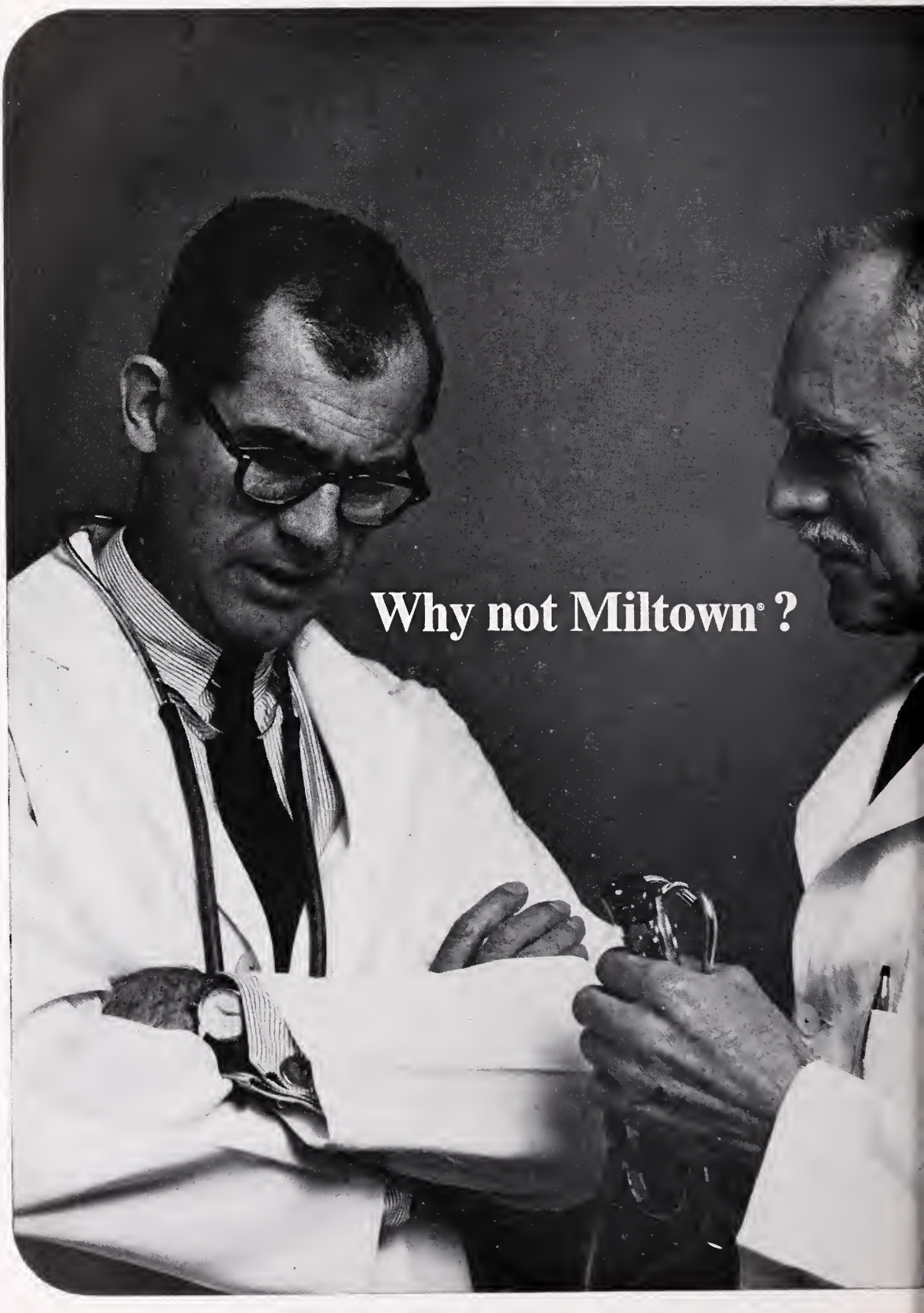
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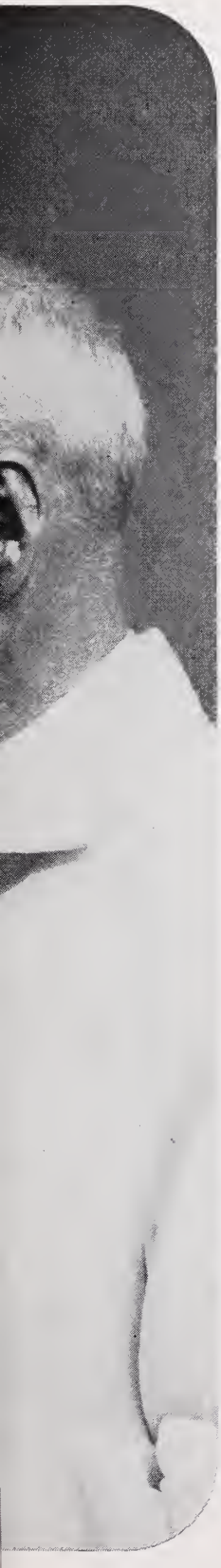
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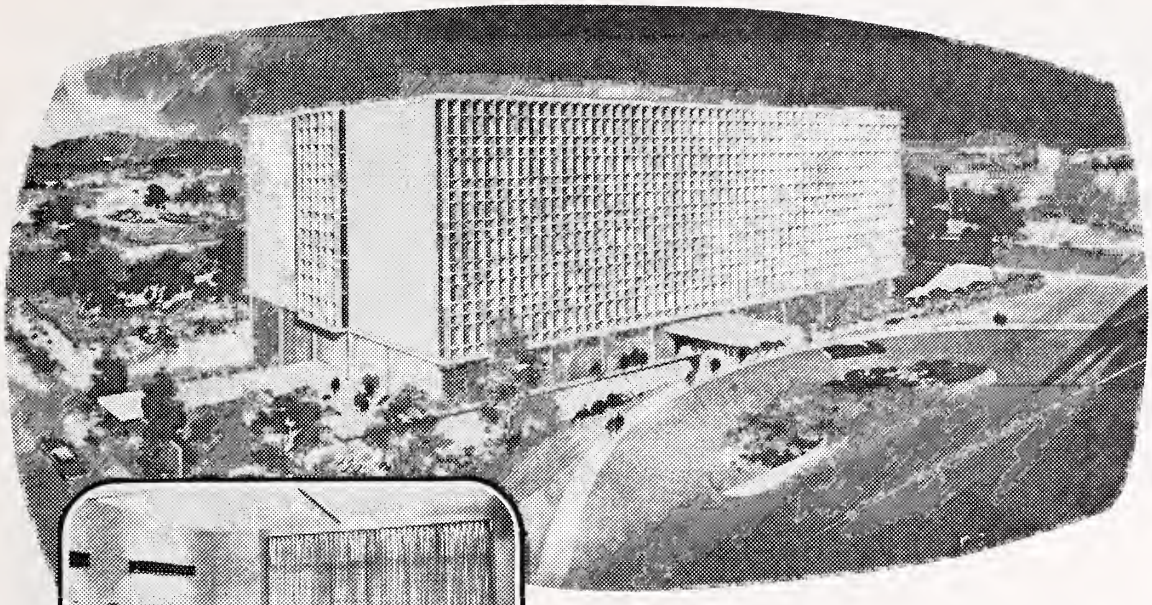
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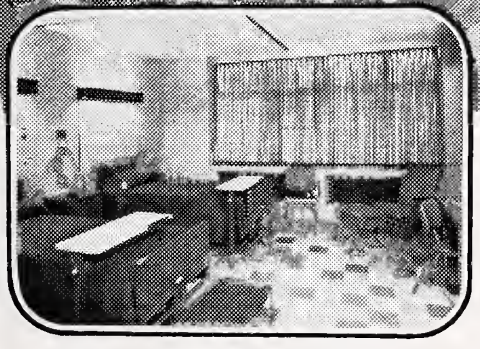
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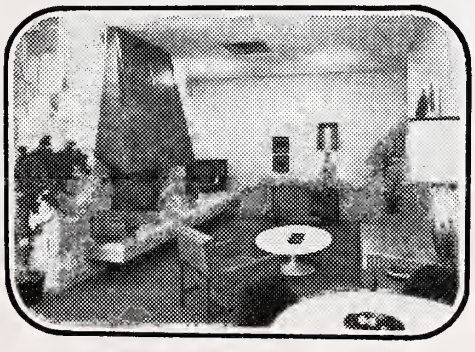
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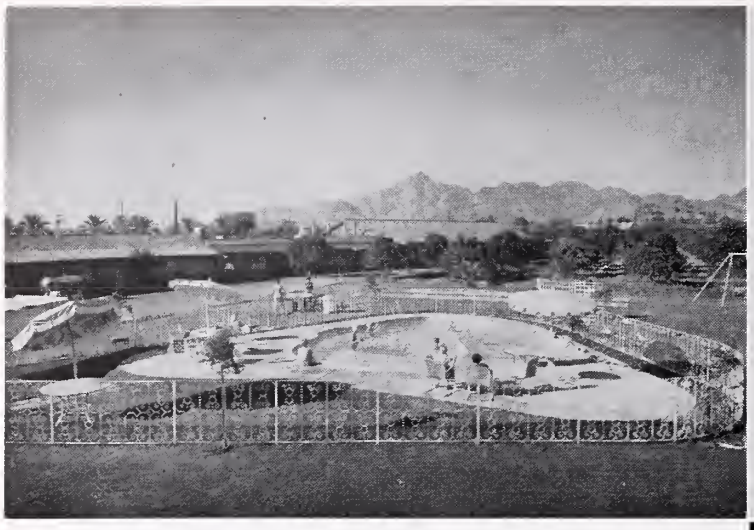


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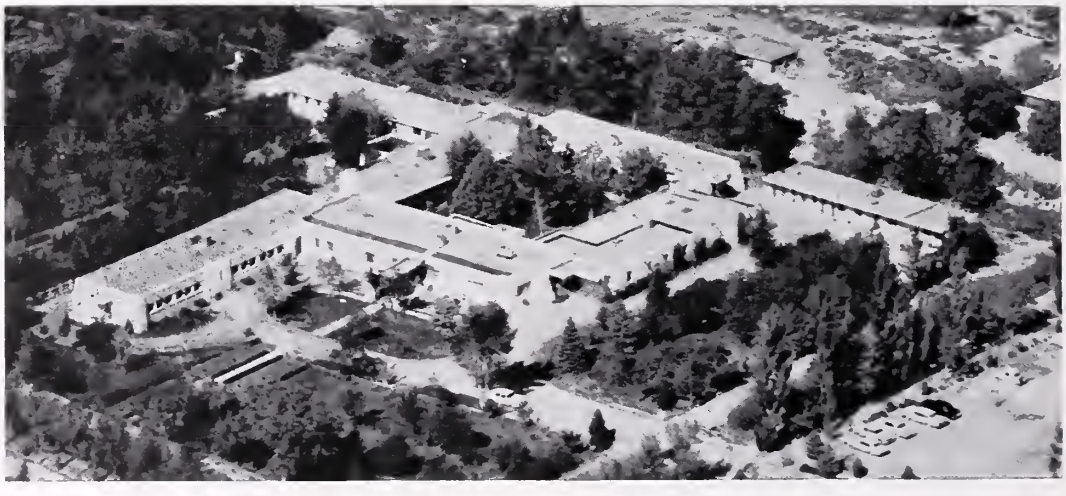
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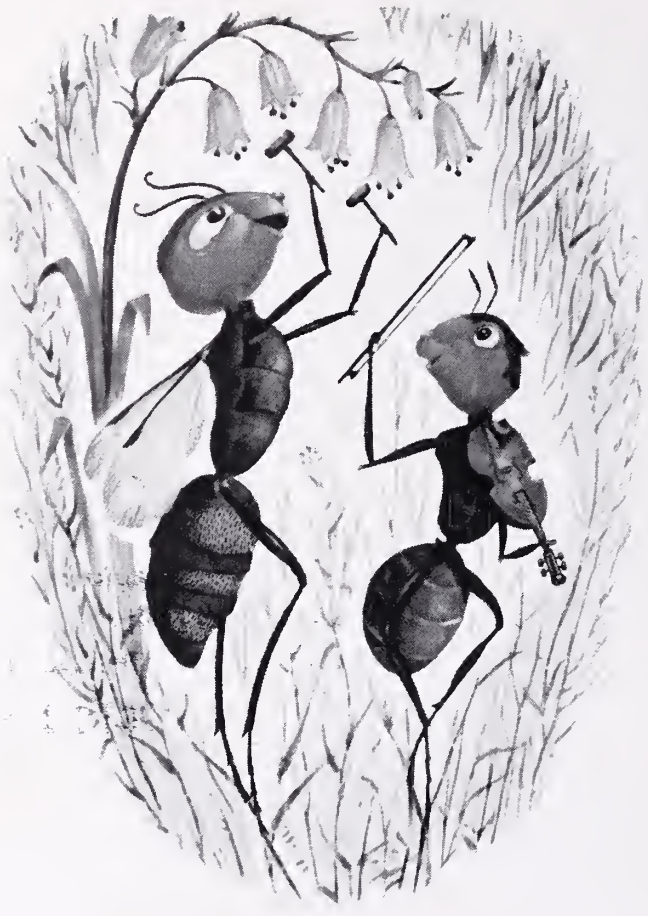
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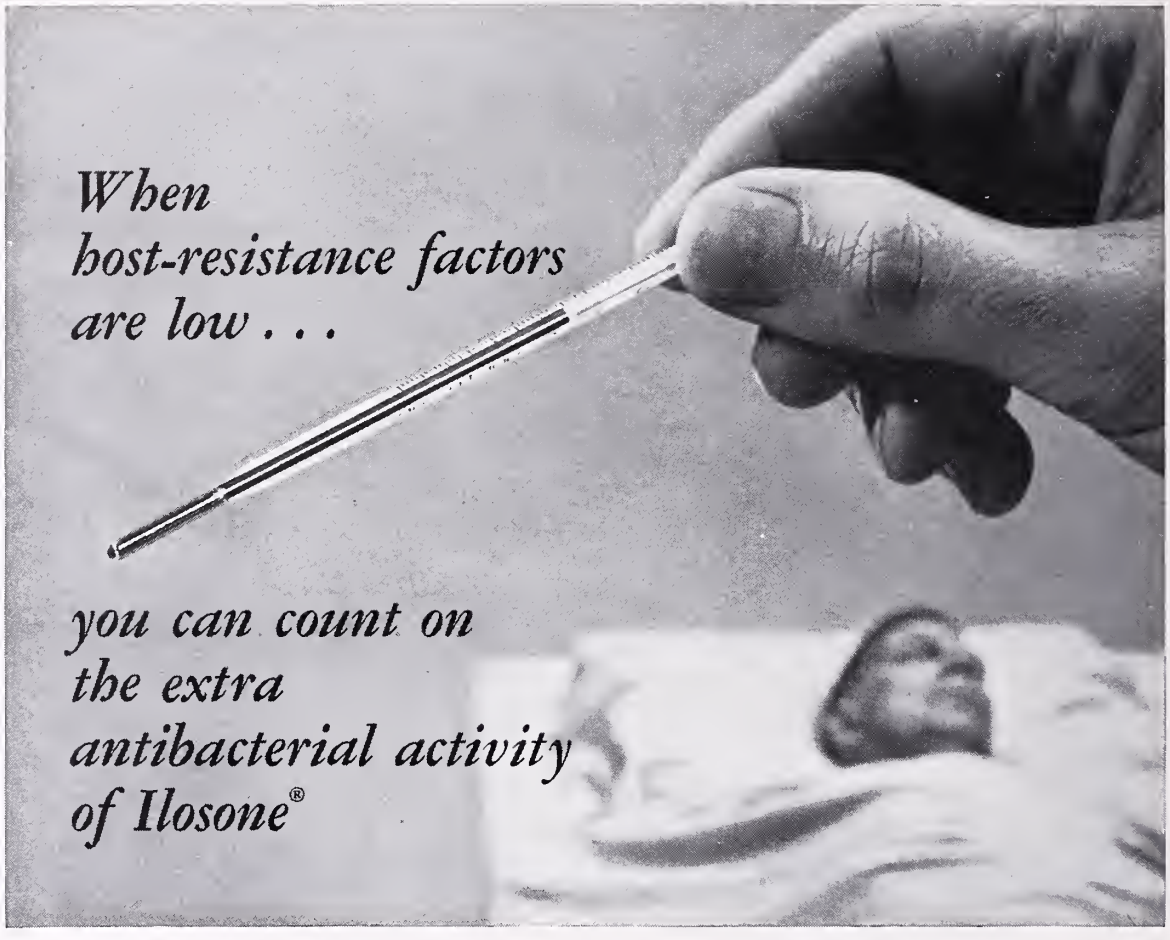
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*Pichler, H.: HNO 4:178-179 (May 28) 1954.

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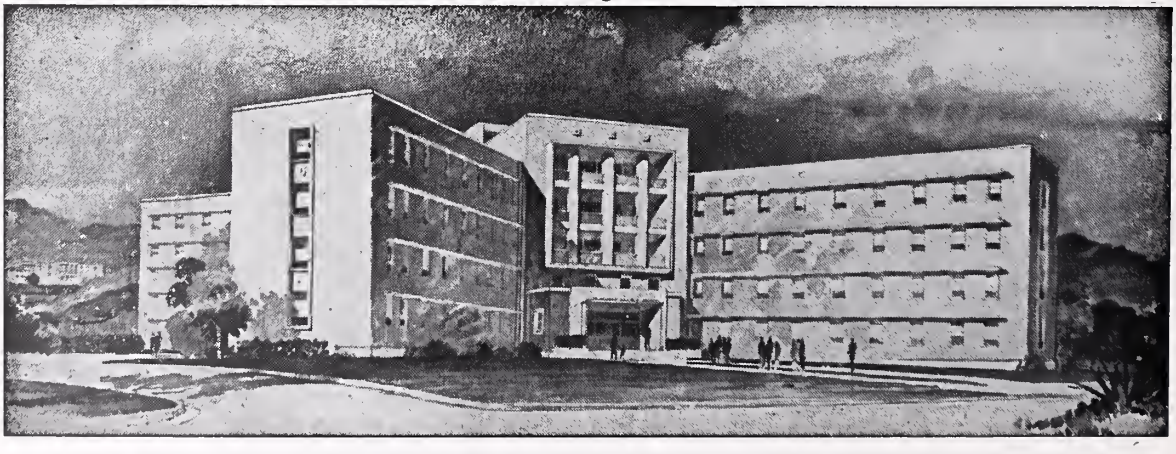
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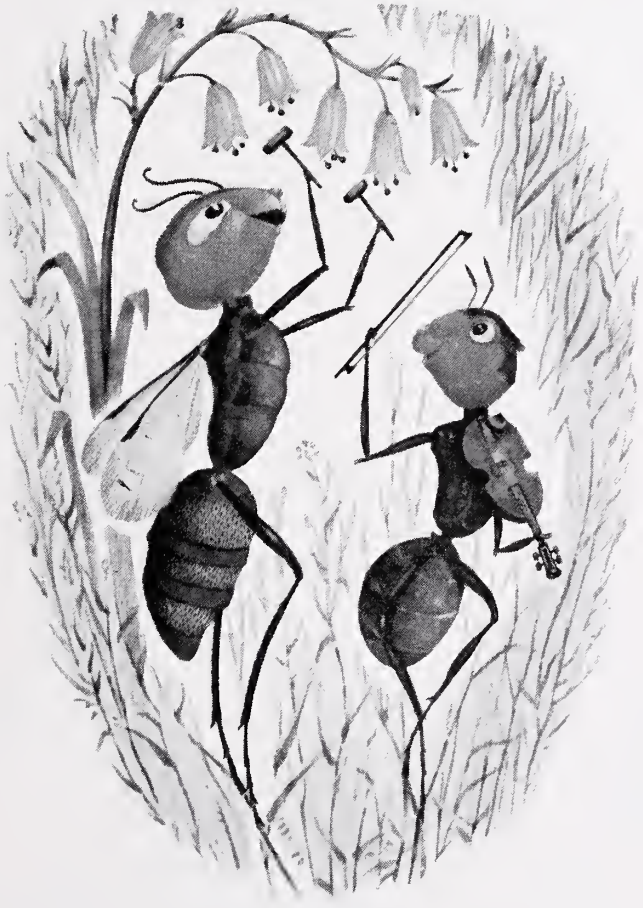
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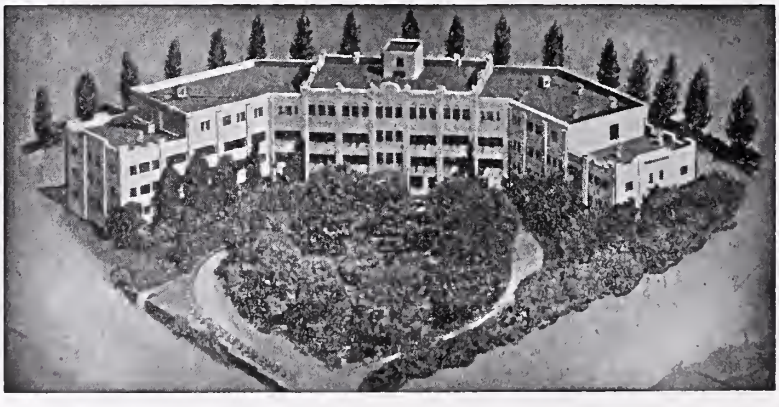
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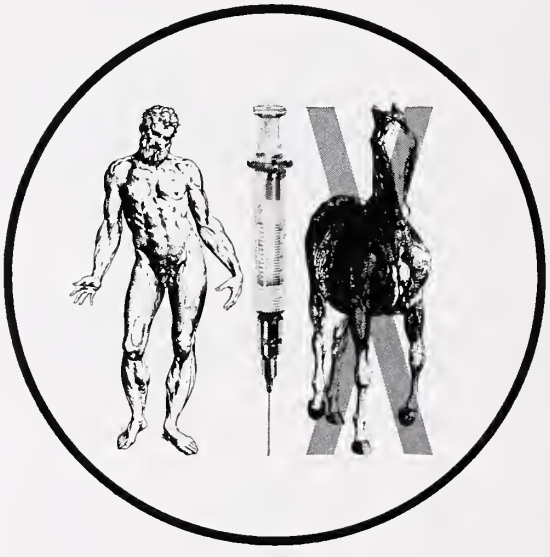
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References: 1. Rubbo, S. D., and Suri, J. C.: Brit. M.J. *2*:79 (July 14) 1963. 2. Rubinstein, H. M.: Am. J. Hyg. *76*:276, 1962. 3. McComb, J. A.: New England J. Med. *270*:175 (Jan. 23) 1964. 4. Effective tetanus protective level established by Sir David Bruce. 5. Editorial: Tetanus Immunization, JAMA *161*:883, 1956.

APHORISMS and MEMORABILIA *

ANDREW M. BABEY, M.D., *Las Cruces, New Mexico*

1. Dysphagia frequently is the first symptom of myasthenia gravis — in 35 of 175 cases in one series (J.A.M.A., *134,* 987, 1947). (pg.3)

2. Dysphagia is a frequent symptom of cerebellopontine angle tumor — 16 of 50 cases in one series (Proc. Staff Meet. Mayo Clin., *25,* 549, 1950). (pg.3)

3. Approximately 20 per cent of patients with hiatus hernia have esophagitis. (pg.8)

4. Approximately 25 per cent of patients with esophagitis have hiatus hernia. (pg.8)

5. Pregnancy is a common cause of esophagitis, particularly when there is pernicious vomiting. Stricture may be well formed early in the puerperium. (pg.8)

6. As a generalization, it can be said that the smaller a hiatus hernia is, the more pain it is likely to produce (and the harder it will be to get a surgeon to accept the patient for hernia repair). (pg.11)

7. Ordinarily, air in the lesser sac passes quickly through the foramen of Winslow, rendering radiologic detection difficult. (pg.23)

8. Achlorhyria is a most difficult diagnosis to establish. When established, it has to be limited by some explanation, such as "achlorhydria of the moment", or "achlorhydria this week". The results of gastric analysis vary greatly from day to day. (pg.26)

9. The radiologist is not able to diagnose gastritis. (pg.31)

10. Dumping syndrome sometimes first appears years after surgery. About four per cent of ulcer patients who have had no operation have dumping syndrome. (pg.35)

11. In spite of all the controversy, it seems fair to make the statement that benign ulcers never become malignant. If it makes you feel better, say that one per cent of gastric cancers were once benign ulcers, only to give you an out for that one questionable case you may see in your lifetime. (pg.35)

12. On the other hand, hyperplastic polyps and the hyperplastic stage of atrophic gastritis are probably premalignant lesions. Pernicious anemia appears to be the only systemic disease which, through its gastritis, predisposes to gastric carcinoma. (pg.35)

13. A personal belief is that cancer of the stomach is almost not curable by any currently

*From: "Pearls in Gastroenterology" by Colonel Eddy Palmer, M.C., USA, Washington, D.C. 1955. Reprinted with permission of Colonel Palmer.

available therapeutic technic. Don't be fooled by the reports from some clinics which seem to say that five year survivals are being observed in 30 per cent of gastric carcinoma patients. Many pathologists class simple polyps as Grade I carcinoma. Bald figures fail to explain about the many cases over which the pathologists disagree. Professional cancerphabia is such that, if there is any suspicion of cancer, the case is listed as cancer. Distinguish carefully between operability, resectability, palliation, survival, cure. (pg.36)

14. If one is to practice honest medicine, an almost superhuman amount of courage is usually necessary when dealing with the patient's family on a case of gastric cancer. There are those who can't believe that the kindest and best treatment for the patient with gastric cancer is to leave him alone. The argument, "Surgery offers the only chance", is the most diabolical in medicine. It seems that in cases of cancer, especially, the doctor has trouble sublimating that subconscious drive to treat himself, rather than his patient. (pg.37)

15. Without surgery, 10-15 per cent of patients with gastric cancer survive five years after the onset of bona fide cancer symptoms. (pg.37)

16. Ulcers are caused by people. (pg.38)

17. Fever occurs in the majority of patients with important gastrointestinal bleeding, regardless of source. May reach 103°. It usually appears within 24 hours. However, fever does not follow experimental ingestion of whole citrated blood. (pg.43)

18. Cirrhotic patient with hematemesis: the most dangerous clinical assumption is that the hemorrhage is originating from varices. Among cirrhotics with hemorrhage: 17 per cent don't have varices at all, 36 per cent of those with varices have other potential bleeding lesions (New England J. Med., *248,* 1057, 1953). (pg.43)

19. It may be very difficult or impossible to tell that there is severe ulcerative colitis by inspecting the exterior of the colon at laparotomy. There may be some hyperemia or spasticity only. (pg.56)

20. There is an idiopathic form of acute ulcerative proctitis which seems far commoner and more important than one might guess from reading the books. It is characterized clinically by rectal bleeding, mild diarrhea, and a sense of incomplete emptying of the bowels. It is likely to clear spontaneously, only to recur at intervals of several or many months. It does not appear to be related to the earliest stage of ulcerative colitis nor can one make abortive ulcerative colitis out of it, the reason being that it may clear so quickly. Sigmoidoscopy shows the picture of the active phase of ulcerative colitis, but the process stops abruptly at the first or second valve or, less frequently, at the rectosigmoid junction. A 10-day course of sulfa will often appear to clear the process completely, with reversion of the rectal mucosa to normal at the end of that time. But no pathogenic organism can be found. (pg.60)

21. A significant proportion of patients with ulcerative colitis give a history of hemorrhoidectomy immediately preceding the first symptoms of ulcerative colitis. (pg.60)

22. Fight everywhere you can the unfortunate professional and lay understanding that amebiasis is almost impossible to diagnose and to cure. These very serious misconceptions grew out of poor understanding of the technic of diagnosis. Once a patient has been told, erroneously, that he has amebiasis, it will be very difficult to correct the error, especially if the original wrong diagnosis was made only after long clinical study. (pg.62)

23. The commonest sin in amebiasis is specific treatment when the specific organism cannot be found. The word "amebiasis" has been dropped too often to neurotic patients with irritable colon. The patient carries it from doctor to doctor, and treatment too often is represcribed. In amebiasis, never treat unless the organism is demonstrated (there is an exception in the case of amebic liver abscess). (pg.62)

24. One of commonest of all ameba misconceptions: the supposed importance of whether a patient is passing cysts or trophozoites. Get this straight: amebae emerge from the colon wall into the lumen as trophozoites. In the fecal stream they encyst. This takes time. If the patient is having diarrhea, encystment cannot take place because the amebae are passed too quickly and because the necessary dehydration of the fecal stream has not taken place. If the stool is formed, the amebae will be passed in the cyst stage. To determine whether a patient is passing trophozoites or cysts is to determine whether he is having diarrhea or formed stools. (pg.62)

25. A propos of the above, the amebiasis patient with diarrhea doesn't necessarily have worse amebic colitis than the one with normal stools. But as far as diagnostic technics are concerned, the consistency of the stool makes a great deal of difference: cysts can be concentrated by flotation technics, but there is no way of concentrating trophozoites except by culture. (pg.62)

26. Although it is true that liquid stools should be examined warm, formed stools may be stored in the ice-box for several days without compromising the diagnosis of amebiasis (as well as other cyst-forming protozoa and intestinal helminths). (pg.62)

27. Amebic colitis is a right-colon disease primarily, and positive sigmoidoscopic findings are unusual. Trans-sigmoidoscopic scrapings of normal appearing mucosa, nevertheless, at times will provide the diagnosis. (pg.62)

28. There is something about liver which is antagonistic to *E. histolytica*. The rarity of amebic abscess of the liver can be explained only on this basis. If you should be carrying experimental amebiasis in dogs, don't feed them liver because it will cure them. (Don't feed them salmon, either, because they will all die of fulminating amebiasis.) (pg.63)

29. For one to four months following successful treatment of amebic colitis, there is often a troublesome irritable colon syndrome. This post-treatment syndrome must be recognized and respected, lest it lead to repeated courses of anti-amebic treatment in people who no longer have amebiasis. Patient may feel that the initial course of treatment has done no good and he may insist on more (Am. J. Trop. Med., *26*, 543, 1946). (pg.63)

30. Pinworm eggs saturate the foments of an infested house. They travel with the air currents. In any orphanage they may be recovered from the tops of picture frames, chandeliers, toy box. The problem of treating the patient is nothing as compared with that of treating the house. (pg.64)

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Impressions of Medicine in the Philippines

C. Pardue Bunch, M.D., *Artesia, New Mexico*

During August of 1964 it was my privilege to be the guest of Dr. Jose Santillan and the Academy of General Practice of the Philippines for a three-day visit. Dr. Ramon Macasaet, President of the Academy, and Dr. Ramon Angeles, Past President, were among those on the Welcoming Committee at the airport on our arrival. An orientation dinner was given for us that evening at which a dozen doctors and their families were present to welcome my wife, my son, George, and my daughters, Charlotte and Winkie, to the Philippines. We were given tours during the next two days of areas within 150 to 200 miles of Manila.

It was our impression that Manila, with more than a million people, is quite a modern city although there is not a great deal of industry in the Philippines. There are many modern buildings, most of which have been built since World War II, during which almost all the buildings were destroyed in Manila. However, when one gets more than 50 miles away from Manila, he is aware that he is in a rather primitive type of rural semi-tropical country. There are very few motor-propelled vehicles, and most of the people travel by horse drawn calesa similar to a two wheel buggy. The agriculture is primarily rice, sugar cane, coconuts and fruits. Rice fields are cultivated by carabao, a type of water buffalo, and by a primitive type of plow. The people live in villages, made up largely of huts of bamboo or grass, built up on stilts to avoid floods. Most of these villages did, however, have an elementary school and a small medical clinic where government doctors provide at least the rudiments of medical care. I was informed that there was about one doctor for every 5000 people in the rural areas of the Philippines, whereas there is one doctor for every 1600 persons in the city of Manila.

People Friendly

The people are very friendly. Eighty-five percent of the 20 million Filipinos belong to the Roman Catholic Church. They are a mixture of Malaysian, Chinese, Indian, Arab and Spanish origins. Since 1898, when the Americans took over the Philippines, the English language has become the principal language. However, today Spanish is still spoken by many and the official language of the country is Tagalog, which is the native language of about one-third of the Philippine population.

Manila Hospitals

During our third day in the Philippines, Dr. Santillan took me to visit the North General Hospital, a government hospital of 450 beds which seemed to be fairly modern, although somewhat overcrowded. This is half the size of the largest hospital in Manila, the Philippine General Hospital, which is the best equipped and most modern hospital in the Philippines. Dr. Ordonio J. Reyes, the Medical Director, received his training in Pittsburgh. He told me that almost all members of the staff had had graduate training in the United States after they had completed their training in one of the Philippine medical schools. The University of the Philippines and the University of Santa Tomas have the two largest medical schools in the Philippines. However, there are five or six private medical schools in various parts of the Philippines also. An article by Dr. J. Z. Bowers of the Rockefeller Foundation in the JAMA Volume 191, Number 2, January 11, 1965, gives an interesting summary on "Medical Students in the Philippines".

At the North General Hospital, Dr. Reyes told us that 90 per cent of the patients were charity patients. They do have a few private rooms which cost about $10 in American money daily and a small number of semi-private rooms that would be about $4 a day American money. This hospital is equipped with a cobalt therapy unit, and it has a Betatron machine on order. I visited the operating rooms and the Obstetrical suite and



Dr. C. Pardue Bunch and family being greeted at Manila Airport, August 12, 1964, by Philippine doctors.

found them to be equipped with modern facilities. In the nursery there were three isolettes, and they had a total of nine prematures under their care at the time with a total of 43 babies in the nursery. This hospital has 67 Residents and 50 or more Specialists on the consulting staff who provide the teaching program there.

Chinese Hospital

Later in the day Dr. Santillan took me to visit the Chinese General Hospital which has been built by the Chinese community. Dr. Santillan received his Intern training at this hospital about 30 years ago. Dr. Tan, Medical Director of this hospital, showed us their fairly well-equipped institution. Thirty-three per cent of their patients are charity patients. They have a School of Nursing with 100 students in each class. At present they have 16 Interns and Residents and he told me they did about 10 to 15 deliveries each day. They are building a new wing which is much more modern than the old part of the hospital. Their rates per room are about the same as those at the North General Hospital except that they did have ward rooms for approximately $2 per day for patients who were able to pay only a small amount. Dr. Tan stated that they were operating in the black at the time.

Mission Hospitals

The third hospital we visited in Manila was the Mary Johnson Hospital, the only mission hospital of the Methodist church in the Philippines. This hospital has 120 beds, was rebuilt about 10 years ago and is more modern than the Chinese General Hospital. However, it is in a slum area of the city near the harbour and 90 per cent of the patients are charity patients. Dr. Catindig, Medical Director, received graduate training in the United States. They have a full-time Philippine Chaplain, and located across the street from the hospital is a Methodist church which provides spiritual and social services for the patients of this area. There are of course several mission hospitals of the Seven Day Adventists, the Mormons, the Baptists, and other denominations in Manila and throughout the Philippines. These are manned largely by Philippine doctors, many of whom received some training in the United States.

Rural Medical Care

I was informed that very few of the doctors care to go back to rural areas to practice. Most of the medical care in these areas is provided by doctors of the government's Public Health Service and is still very limited and inadequate. There is still need for a great many more general practitioners, both in Manila and the outlying areas of the Philippines.

Summary

The physicians are very friendy to the United States and follow the American system of medicine as far as possible. Their standards of medical care are improving and with cooperation from our country, they can certainly come up to American standards in the forseeable future. I was invited to give a lecture on "Hypnosis in Obstetrics, Smoking, and Weight Reduction" and another lecture on "Dermatology in General Practime". Copies of the "Family Physician" published quarterly by the Philippine Academy of General Practice, and "M.D.", an independent journal, contained articles by Philippine and American doctors and showed a real effort to improve standards of medical care and education in this growing, developing, friendly Republic.



N. M. OFFICERS—New officers of the New Mexico Medical Society, elected at the annual meeting in Santa Fe, May 9-15, 1965, are Dr. Robert P. Beaudette, second from left, Raton, President, Dr. Thomas L. Carr, second from right, Albuquerque, President-Elect, Dr. Emmit Jennings, left, Roswell, Vice-President, and Dr. John D. Abrums, Albuquerque, Secretary-Treasurer.

Dr. Beaudette, Raton, Elected President N.M. Medical Society

Dr. Robert P. Beaudette, Raton, was elected President of the New Mexico Medical Society at its 83rd annual meeting, held in Santa Fe, May 9-5, in conjunction with the 13th Biennial Rocky Mountain Medical Conference.

Other new officers are Dr. Thomas L. Carr, Albuquerque, President-Elect; Dr. Emmit Jennings, Roswell, Vice-President; and Dr. John D. Abrums, Albuquerque, Secretary-Treasurer. Dr. Hugh B. Woodward, Albuquerque, retiring Secretary-Treasurer, was elected Speaker of the House of Delegates, Dr. Ronald V. Dorn, Albuquerque, Vice-Speaker, and Dr. John Smoker, Raton, Dr. Harry Ellis, Santa Fe, and Dr. Earl Flanagan, Carlsbad, Councilors. Clovis will be the site of the 1965 Interim meeting and Albuquerque the location for the 1966 annual meeting.

Dr. Woodward was presented the Fifth Annual A. H. Robins Award for Community Service.

Dr. Woodward, who served as Secretary-Treasurer for the State Society from 1962 to 1965, is widely known for his work as Coordinator of the State-wide Sabin Oral Vaccine Campaign in 1962-63 when he was Chairman of the Society's Public Health Committee.

He is Vice-President of the Albuquerque Community Council. He has been a United Fund Director and Vice-President, a member of the Bernalillo County Indian Hospital Executive Committee, and Coordinator of the New Mexico Tuberculosis Council. In 1964 he served as Chairman of the Building Committee of St. Joseph Hospital, where he has been Chief-of-Staff and a member of the Executive Committee, and as Chairman of the Health Sub-Committee of the Community Survey. He is a Past President of the New Mexico Society of Internal Medicine. Dr. Woodward received his M.D. from Jefferson College of Philadelphia and began the practice of medicine

in Albuquerque in 1954.

Some of the action taken by the House of Delegates is as follows:

That members of the New Mexico Medical Society be permitted to communicate with Doctors of Osteopathy when the medical welfare of the patient requires it for the acquisition of clinical data only, recognizing that this is not to be construed as a medical consultation.

That the New Mexico Department of Public Health administer the Kerr-Mills Law rather than the New Mexico Department of Public Welfare.

That the Mental Health Committee develop a program with professional assistance from the University of New Mexico Medical School to sponsor psychiatric seminars for non-psychiatric physicians.

That the Liaison Committee to the UNM Medical School investigate the possibility of televising selected clinics and regularly scheduled Basic Science Series and/or other methods of continuing medical education.

The new President, Dr. Beaudette, was born in Burlington, Vermont, and received his B.S. from Holy Cross College in Worcester, Massachusetts, and his M.D. from the University of Vermont School of Medicine. He began the practice of medicine in Raton in 1949.

He is certified by the American Board of Opthalmology. He is a member of the Board of Directors of Blue Shield and has served as President of the Raton Chamber of Commerce. The Beaudettes are the parents of five boys and three girls.



ROBINS AWARD—Dr. Hugh B. Woodward, left, Albuquerque, is presented the Fifth Annual A. H. Robins Award for Community Service by Dr. Omar Legant, Albuquerque, outgoing President of the New Mexico Medical Society, at the organization's 83rd annual meeting in Santa Fe, May 9-15, 1965.

Everyone knows Mothers deliver their children once in the hospital and forever after in the station wagon. — *quoted from "The Battered Mother Syndrome" by Margaret Smith, Coastal Bend Medicine.*

A Novel Method of Enhancing Antibody Production

Leighton S. King, Research Associate
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Introduction

Recovery from infectuous diseases of either viral or bacterial origin depends very largely on the ability of the patient to attain levels of natural resistance adequate to overcome the pathogenic invader. This is most imperative when the infecting agent is a bacteria that is antibiotic resistant or a virus where antibiotics are equally ineffective. The formation of specific antibodies during the immune response to antigenic challenge is one of the most important means of achieving resistance to specific invaders.

In patients with advanced malignancies the ability to produce antibodies has in many cases been noted to be greatly impaired. This, in turn, renders many such individuals vulnerable to serious infection. Restoration of natural resistance to the growth of malignant cells is also very likely a major factor in long-time survival of patients treated by the accepted surgical, radiological, and chemical methods, as well as in those rare cases of spontaneous remission.

Animal experiments indicate the possibility that latent viral infections become established and remain dormant until hormone, radiation, or chemical stimulation triggers them into activity. This, in turn, can result in the induction of various pathogenic states, including malignancy.[1] If a highly resistant state, including the capability of a strong immune response, can be attained it is highly possible that the virus may be inactivated and the diseased condition prevented.[2,3] This presumes that the viral agent is not one that was acquired during the first few days after birth and to which the host is immunologically tolerant or that this tolerance can be reversed.

Much work has been done with adjuvants to stimulate increased antibody production. The most effective of these is that of Freund, which consists of an emulsifying agent, mineral oil, saline solution, and killed mycobacteria emulsified and mixed with the antigen prior to injection. Antibody production is increased, but sterile abscesses at the site of the injection frequently occur due to the killed bacteria. This is particularly objectionable when multiple injections are required. Injection of the adjuvant separate from the antigen or after an infection has already started has no appreciable effect on antibody production. The conventional adjuvant is therefore of very little value in combating an established infection.

Purpose

The purpose of this investigation was to determine how antibody formation could be increased from a given antigenic challenge without incurring the objectionable features of conventional adjuvant.

Material and Methods

Swiss mice* of mixed sex and approximately nine weeks of age were used as test animals. They were housed in groups of 40 in stainless steel cages containing wood shavings for bedding ma-

*D.A.L. Swiss mice — Diablo Laboratories, Berkeley, California.

terial. Access to water and food consisting of three parts of oats and one part of Calf Manna** was available at all times. The cages were kept in an air-conditioned room at 78°F. Sterile sheep cells in Alsever's solution were employed as the antigen which was administered subcutaneously in 0.2-ml amounts on four occasions at weekly intervals.

In order to attain maximum levels of antibody production by known methods, 0.2 ml of Freund's adjuvant was mixed with 0.2 ml of sheep cells for each weekly injection of each mouse in one group. Three groups of mice had two per cent of Viokase*** by weight mixed with their food during the test period. One of these groups received no sheep cell injections; the second received sheep cells mixed with Freund's adjuvant; and the third received sheep cells. A fifth group fed on nonsupplemented food received adjuvant injections only without sheep cells. A sixth group on nonsupplemented food was used as controls.

At the end of five weeks on this regime, each group was given light ether anesthesia and bled to death by exposing and severing the subclavian vein. The blood was pooled for each group, allowed to coagulate, and centrifuged to obtain the clear serum. Sheep cell hemolysin content of the serum from each group was then determined.

Results

The following results were obtained from the foregoing experiments:

1. Freund's adjuvant mixed in equal parts with sheep cells before injecting gave a 250 per cent increase in antibody formation over controls receiving only sheep cells.
2. No sheep cell antibody was present in the serum from mice which did not receive sheep cell antigen regardless of diet or adjuvant administered.
3. Mice fed with food containing two per cent Viokase and challenged with sheep cell antigen formed 260 per cent more antibodies than controls receiving the same antigen but on nonsupplemented food.
4. The combined effect of adjuvant mixed with antigen and Viokase additive in the food did not stimulate antibody levels above that obtained by either method alone.
5. Sterile abscesses at the site of the injection of Freund's adjuvant were prevalent.
6. Three groups of mice fed with two per cent Viokase added to their food for two years failed to manifest any unusual symptoms other than weighing approximately 10 per cent less than controls on the unsupplemented diet.

Discussion

In the prescreening effort which preceded these experiments, Viokase was found to be more effective than raw pancreas, which first gave promise of antibody stimulation. A considerable number of materials were tried, but Viokase was far more effective and less harmful to the animals. The reduced weight of animals receiving Viokase as compared to controls on nonsupplemented food is probably due to the somewhat unpalatable taste of Viokase, which results in less food being consumed. This effect has been noted by Ershoff and others[4,5,6] who also reported hypertrophy of the submaxillary glands of rats receiving appreciable amounts of Viokase mixed with certain rations.

The failure of simultaneously administered Viokase and Freund's adjuvant with antigen to produce more antibody than either alone with antigen was likely due to the maximum capacity of the animal to produce antibody being reached.

It is not known at this time what component of Viokase is the active principle, but tryspin, amylase, lipase, carboxy peptidase, and other pancreatic enzymes are present. Additional work currently in process should provide a better understanding of the active stimulant to antibody production.

An extensive feeding experiment in which an oncogenic virus was used in C3H mice, which induces adeno carcinoma in 80 to 90 per cent of the adult females, gave the encouraging results of complete protection for the Viokase-fed females for the entire 24-month test period as compared to 85 per cent deaths from mammary tumors in the non-Viokase-fed females. The details of this experiment are in the process of publication.

Summary

The inclusion of two per cent of Viokase in the

**Calf Manna: High protein - vitamin - mineral animal food manufactured by Albers Milling Company.
***Viokase Powder, 4 N.F. Pancreatin, VioBin Corporation, Monticello, Illinois (whole dried pancreas, powdered and activated).

food of Swiss mice enables them to produce 260 per cent more antibodies to antigenic challenge with sheep cells than in controls receiving the same antigenic challenge.

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Dr. J. J. Stein To Speak At S.W. Medical Ass'n. Meet



Dr. Justin J. Stein, Professor of Radiology and Chief of the Radiotherapy Division of the University of California at Los Angeles School of Medicine, will be one of the Faculty members at the 47th annual meeting of the Southwestern Medical Association in El Paso, November 4-6, 1965, Dr. A. Robert Nering of El Paso, Program Chairman, has announced.

The three-day program will be presented by the University of California at Los Angeles School of Medicine under the direction of Dr. Charles R. Smart, Assistant Professor of Surgery in Residence and Assistant Director of Postgraduate Medical Education.

The general subject for the meeting will be Chest Diseases.

Dr. Stein will speak on "Results of Irradiation Therapy in Carcinoma of the Lung", "Modern Management of the Lymphomas" and will moderate a panel in a discussion of "The Psychodynamics of Respiratory Behavior", "Management of Emphysema and Bronchietasis" and "Management of Mediastinal Tumors".

A graduate of Baylor University College of Medicine, Dr. Stein is President of the American Radium Society, is a member and Past President of the California State Board of Medical Examiners, and is Chairman of the Governor's Emergency Medical Advisory Committee to the California Disaster Office. He is Director of the California Institute for Cancer Research.

Dr. Stein is a Diplomate of the American Board of Radiology, the Pan American Medical Association, and the International Board of Surgery. He is a Fellow in the American College of Surgeons, the Royal Society of Medicine, the American College of Radiology, for which he was also Chairman of its Commission on Cancer, and a Fellow of the International College of Surgeons, of which he is Vice-Regent. He is the author of 77 scientific articles.

The Southwestern meeting will have its headquarters in the Sheraton El Paso Motor Inn in El Paso. Dr. Gordon Black of El Paso is General Chairman. Dr. Clement C. Boehler, El Paso, is President.

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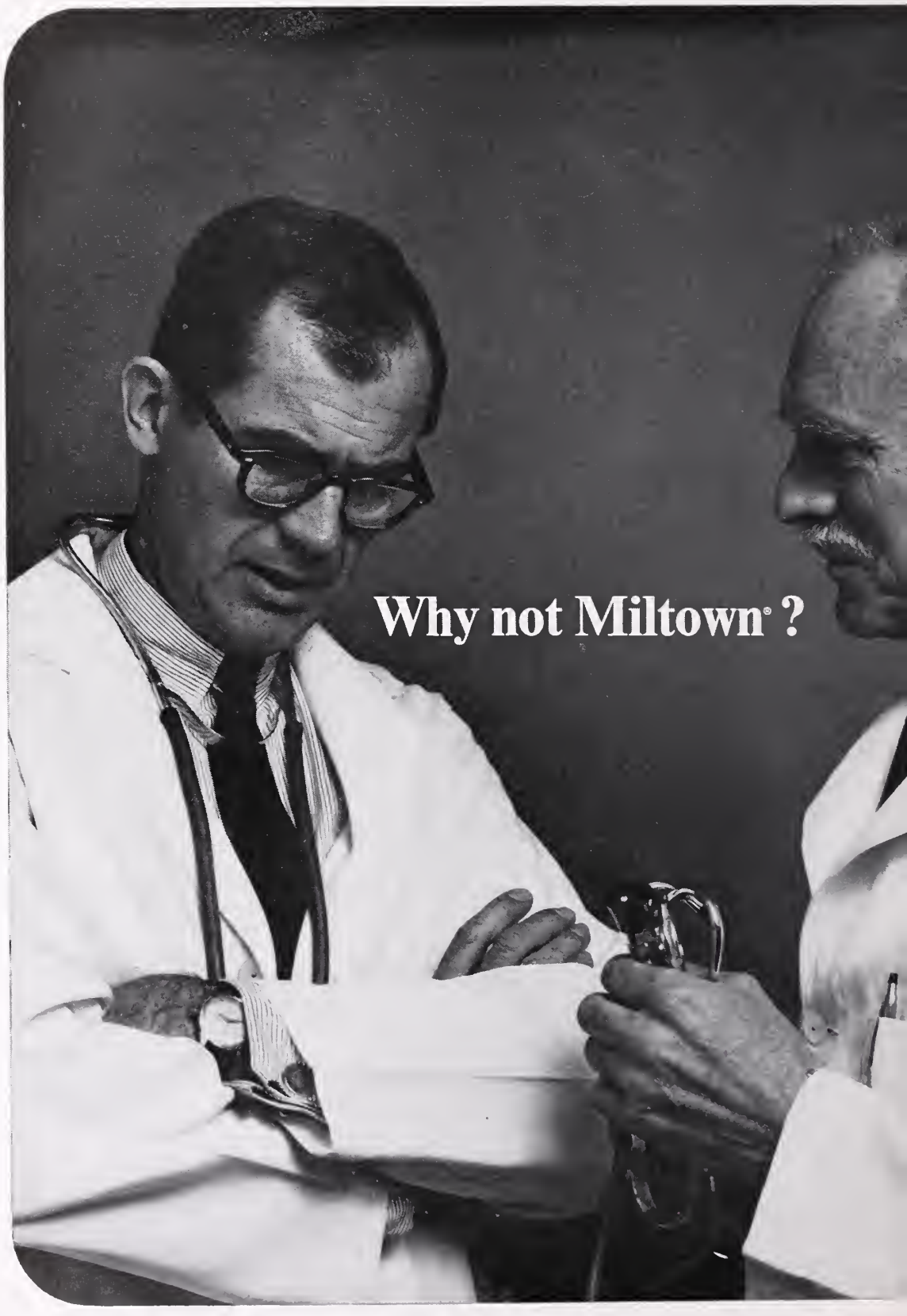
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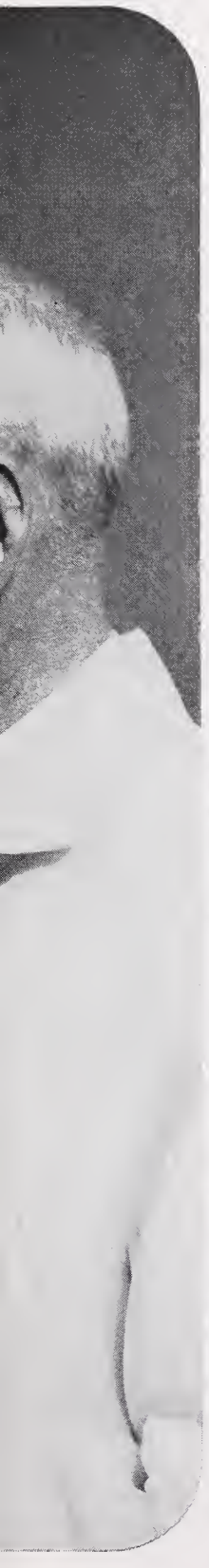
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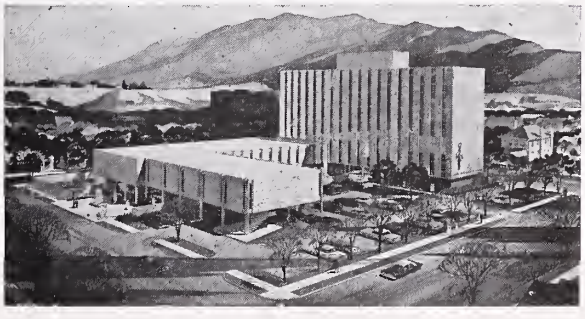
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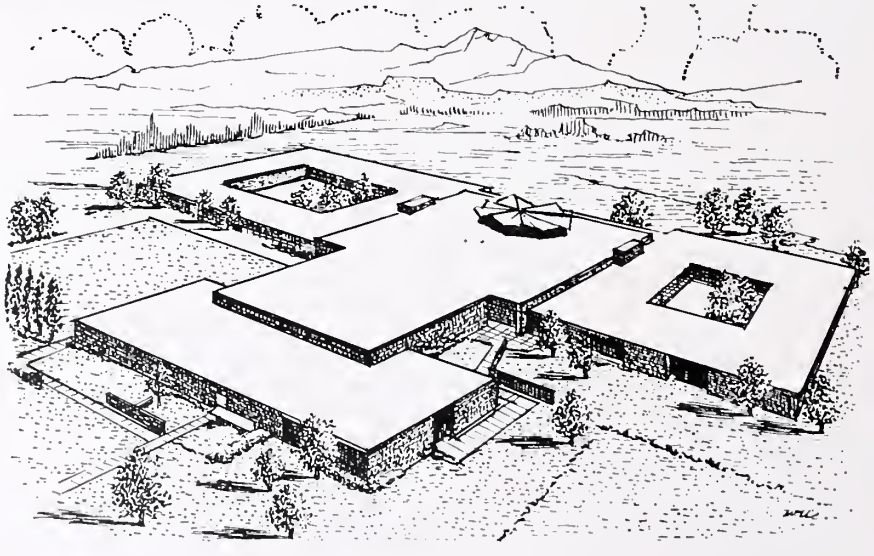
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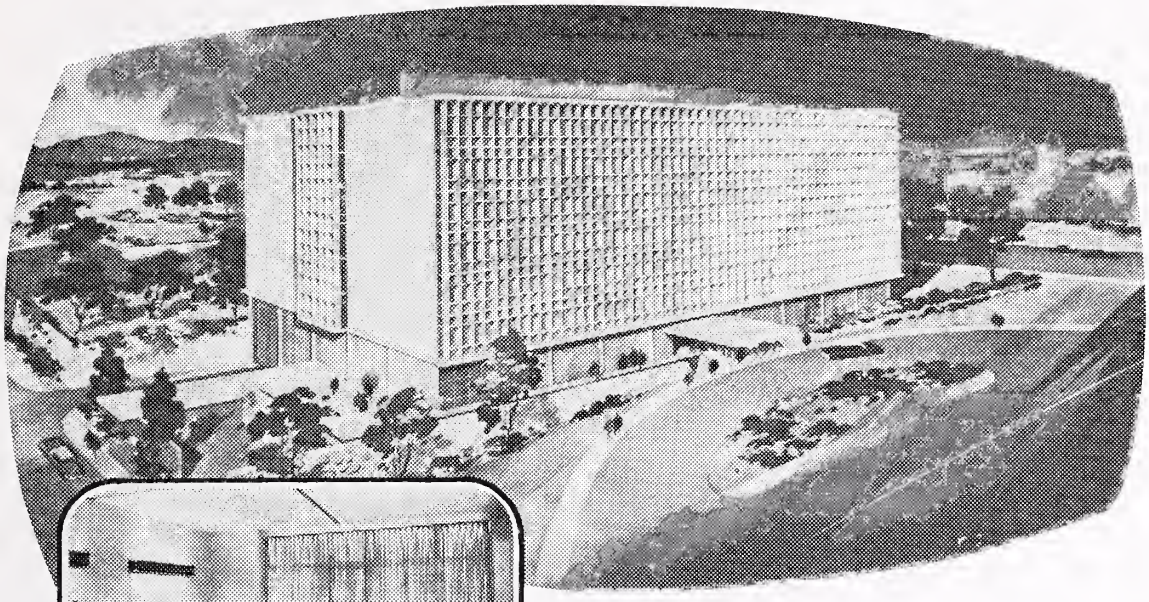


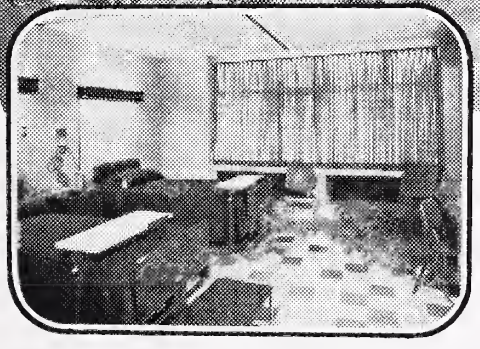
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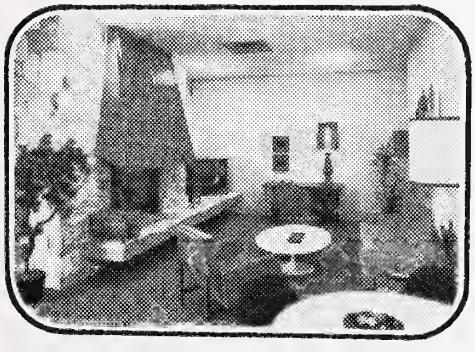
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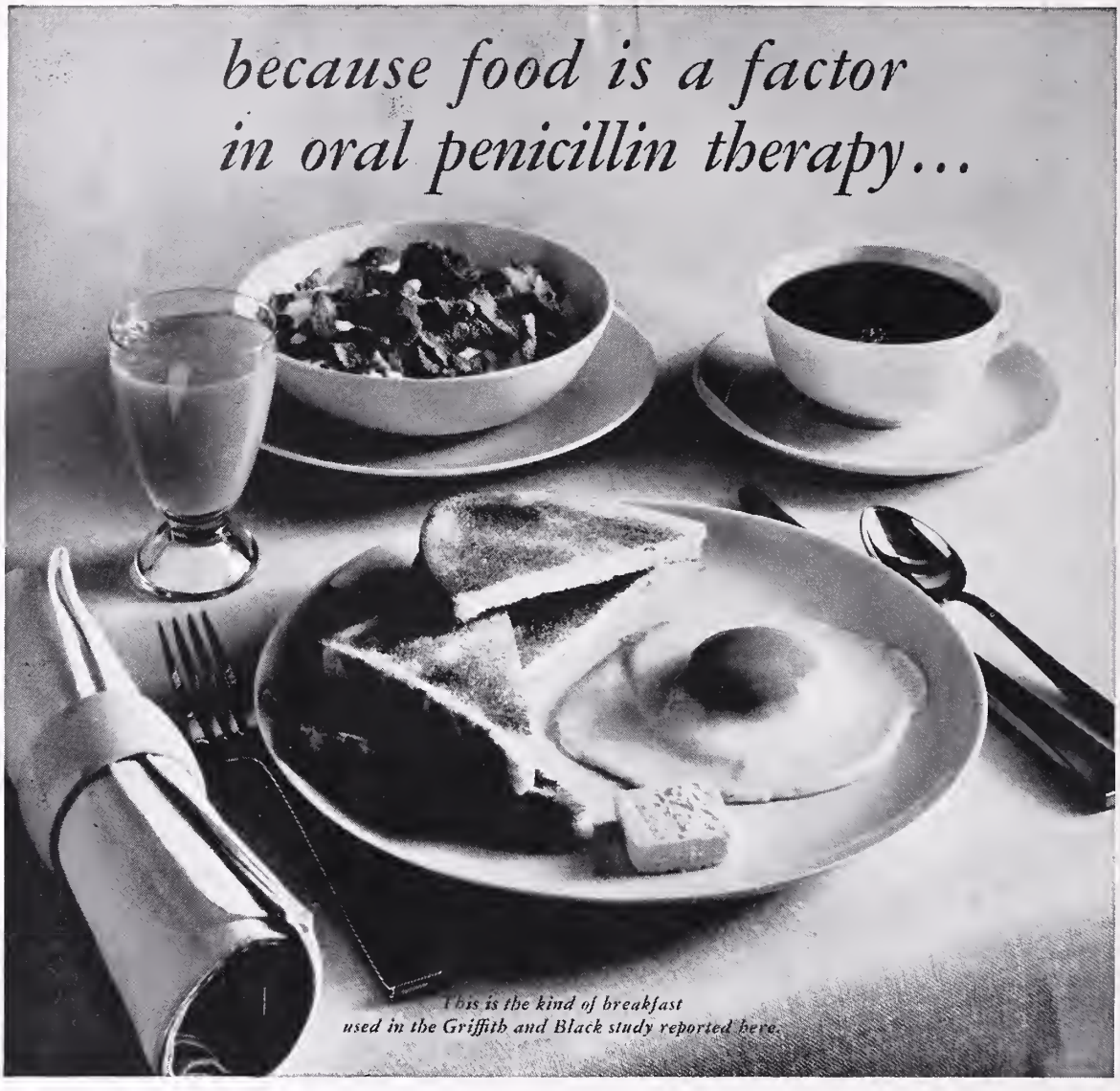
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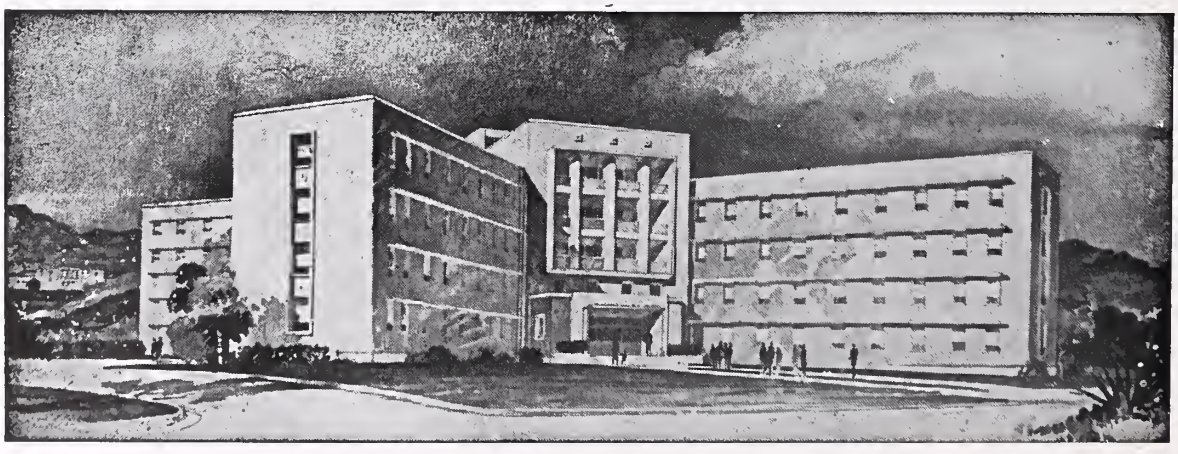
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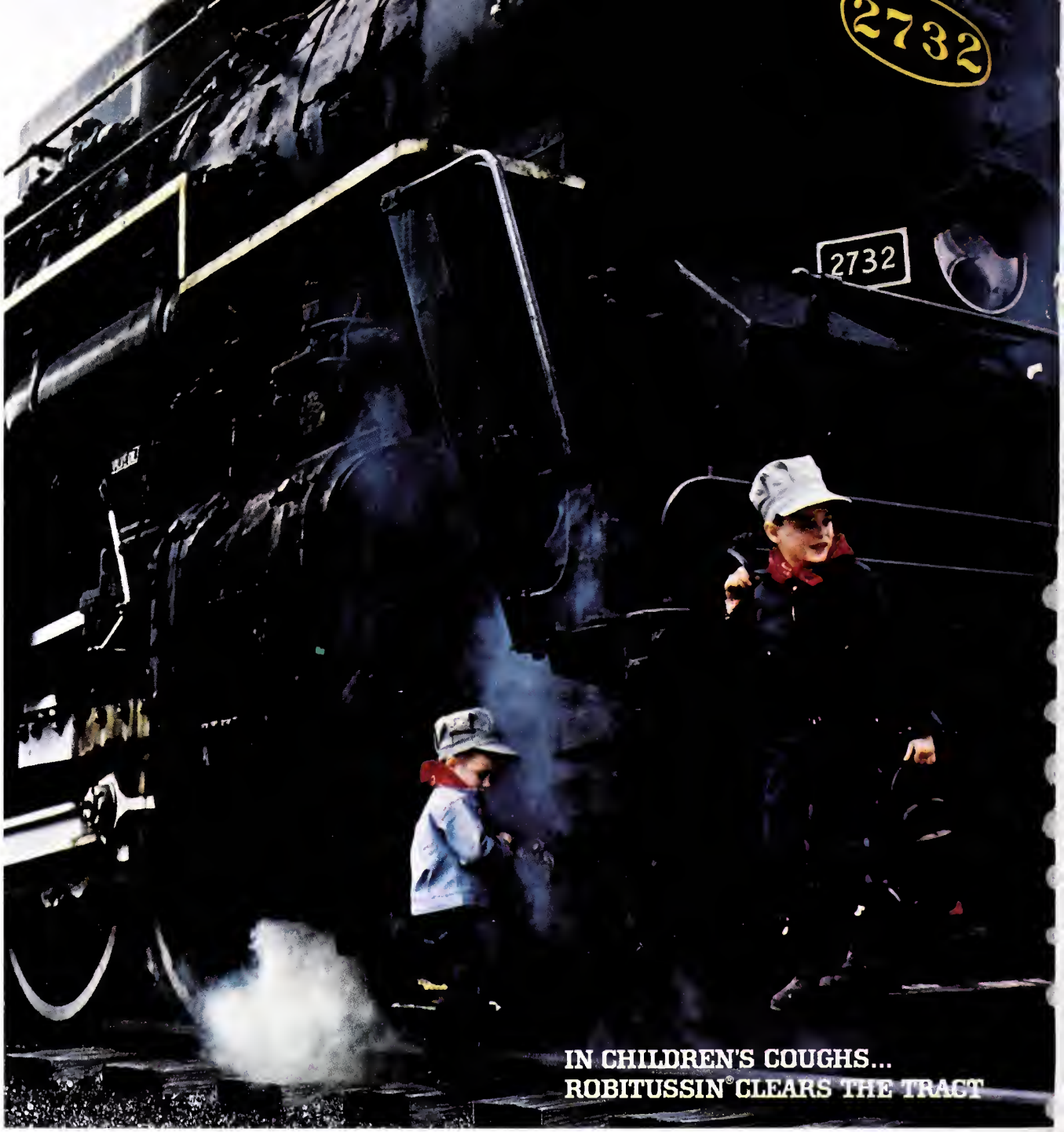


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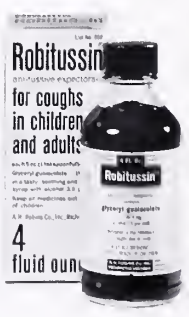
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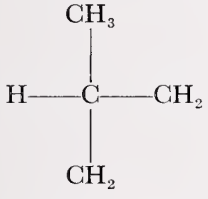
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Intramuscular Dextro-Amphetamine Sulfate in the Treatment of Intractable Obesity and Its Complications*

David B. Gilbert, M.D.**, *Payson, Arizona*

The Drug

The medication used in this study was dextro-amphetamine sulfate in sterile water for injection. Chemically it is the dextro-isomer of d, 1-amphetamine sulfate, commonly known as Benzedrine, with the chemical name of d, beta-phenyl, isopropylamine and the structural formula:



Other chemical names are d-alpha-methylphenethylamine and d-2-amino-1-phenylpropane. The preparation used contained 20 mg of drug per cc in the form of the neutral sulfate.[1]

Review of the Literature

Dextro-amphetamine sulfate has more central nervous system stimulating effect than the racemic form. The usual use for this preparation has been in doses not to exceed 5 mg intramuscularly or intravenously for stimulation of the sensorium in certain depressive states, postencephalitic Parkinsonism, and in narcolepsy. It is classified as a sympathomimetic chemical which imitates the results of sympathetic nervous impulses. This compound differs from the other sympathomimetic substances, epinephrine and ephedrine, in that it has a greater effect in stimulating the central nervous system while the others in this group have a greater peripheral and cardiovascular result. Schulte reports the drug to be the most active as a CNS selective stimulant with the widest margin of safety among 75 adrenergic compounds.[2] This compound has no effect on respiration or body temperature, and Emerson found the effects on the basal metabolic rate to be varied and concluded that the drug had no significant effect on the rate.[4,5,6,7]

Dextro-amphetamine has no effect on the following body chemistries: hemoglobin, red blood count, white blood count, blood proteins, blood sugar or acid-base balance.[7,8,11,12,14]

It has very little effect on pulse, blood pressure, coronary arteries, cardiac output, blood flow, or the electrocardiogram.[7,9,14,16,17]

Galvin and McGavack, Livingston, Rosenberg, Finch, Ferguson and others have found that hypertension is almost never aggravated with the drug, but usually is improved with use of dextro-amphetamine sulfate for the treatment of obesity.[6,9,33,35,36]

The drug's effect on the gastrointestinal tract is not significant and does not account for the anorexic effect which appears to be almost totally cerebral in origin.[8,10,18,19,20,21,22,23]

Effect on the gallbladder is insignificant while urinary sugar and albumin are unaffected also.[8,18,24,25,26]

Dolger and Osserman found that not only are blood sugar levels not increased, but the glucose tolerance of obese diabetics was usually improved when this drug was used.[15]

The human uterus reacts to the drug with increase in muscle tone, but with diminution of the contraction. Large intramuscular doses have

*Read at Regular Medical Staff Meeting, Payson Clinic-Hospital, September 30, 1964.

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not been observed to have any deleterious effect on gestation at any time during the 40-week period, and sex hormone secretion is unaffected while libido remains normal with the use of this drug.[27,28,29] Finch, Coopersmith, and Howell found the drug to be safe and valuable in treating obesity during pregnancy.[6,33,34]

Weiss and Laties state that the amphetamines enhance human performance without a single system paying any price for it.[30]

Leake states that the mechanism of effect is with energy changes altering the usual activity of the cell, by augmenting the level of cellular activity, especially those associated with the sympathetic nervous system.[31]

The drug is 43 per cent excreted in the urine in 48 hours, and inactivation is accomplished in the liver.[13]

Dextro-amphetamine has the greatest margin of safety among 75 sympathomimetic amines tested. The minimal lethal dose is placed at 80 times the therapeutic dose, while amphetamine sulfate has been described as having a lethal dosage of from 100 to 1000 times the average therapeutic dose.[2,3] Injectable dextro-amphetamine side effects have been described as excessive excitement, headache, insomnia and loquaciousness. (This investigator has not found it to aggravate hypertension or angina pectoris, but uncontrolled hyperthyroidism and Raynaud's disease are definite contraindications, as well as marked agitation or anxiety, and hypersensitivity to sympathomimetic agents.)

Dextro-amphetamine sulfate *is not addicting*, since those who have abused the drug do not experience any kind of physical dependence or withdrawal syndrome upon discontinuance.[1]

Some emotionally unstable and frustrated persons who for a means of better facing reality and for the mood elevation properties, may through the illegal and indiscriminate use of the amphetamines, induce serious psychotoxic reactions. The psychological dependency induced by excessive levels of the amphetamine is no more serious than that encountered with the equally potent drug, caffeine, found in the great American habit of the coffee-break.[31]

Amphetamines have never been included in the official listing of addicting drugs put out by the World Health Organization.

Seevers states that the majority of the abusers have been abnormal individuals psychologically, and concludes that instances of drug abuse involves problems with the subject and not with the drug. He further points out that no CNS stimulant is capable of producing addiction and that the depressants are abused to a much greater extent than stimulants, since the latter will eventually lead to unpleasant and unbearable hyperexcitability.[32]

The history of the prescribed use, and discontinuance of use, of dextro-amphetamine sulfate over millions of patients has been free of serious problems and marked discomfort.

Leake reports that dextro-amphetamine sulfate may be used almost indefinitely in therapeutic doses without untoward effect.[31]

The use of this drug in teen-age groups is particularly safe, also.[37,38,39,40]

Other uses of oral dextro-amphetamine are in depressive states, Parkinsonism, behaviour problems in children, enuresis, and epilepsy.

The Study

Patient's Name	Sex	Drug Use Period	Average Daily Dosage	Age	Starting Weight	End Weight	Weight Loss
P. B.	F	3 months	1½ cc	23	164	153	9 lbs.
I. H.	F	3 months	2½ cc	67	198	174	24 lbs.
D. S.	F	3 months	2 cc	42	331	301	30 lbs.
A. G.	F	3 months	2 cc	59	180	149	39 lbs.
G. D.	F	3 months	1½ cc	45	251	245	6 lbs.
E. C.	F	1 month	1 cc		215	198	17 lbs.
M. C.	F	1 month	2 cc	14	168	152	16 lbs.
G. C.	F	1 month	2 cc	40	205	195	10 lbs.
L. K.	M	1 month	2 cc	64	200	188	12 lbs.
L. W.	F	2 months	2 cc	58	226	206	20 lbs.
W. D.	M	2 months	2 cc	56	186	179	7 lbs.
C. G.	F	2 months	1½ cc	59	163	148	15 lbs.
R. O.	M	2 months	2 cc	55	209	200	9 lbs.
N. T.	F	2 months	2 cc	37	176	162	14 lbs.
M. D.	F	2 months	1½ cc	67	182	175	7 lbs.
J. W.	M	2 weeks	3 cc	16	270	250	20 lbs.
V. W.	F	4 months	2 cc	56	180	165	15 lbs.
H. H.	F	4 months	3 cc	42	205	193	12 lbs.
E. M.	M	3 months	2 cc	67	181	155	26 lbs.
R. N.	M	5 months	2 cc	23	270	225	45 lbs.
F. E.	F	6 months	2 cc	48	148	128	20 lbs.
A. L.	F	6 months	2 cc	47	196	177	19 lbs.
E. S.	F	6 months	2½ cc	23	165	136	29 lbs.
S. S.	F	7 months	1½ cc	24	189	158	31 lbs.
J. H.	F	7 months	3 cc	33	189	152	37 lbs.
M. O.	F	14 months	2 cc	46	230	200	30 lbs.
M. J.	F	11 months	2 cc	38	286	192	94 lbs.
V. M.	F	1 year	2 cc	43	235	170	65 lbs.
R. P.	M	1 year	2½ cc	43	220	175	45 lbs.
B. R.	F	1 year	2 cc	58	190	157	33 lbs.
J. S.	M	1 year	5 cc	49	238	179	59 lbs.
R. C.	F	1½ years	2 cc	56	180	146	34 lbs.
V. T.	F	2 years	2 cc	57	180	154	26 lbs.
C. P.	F	3 years	3 cc	32	215	178	37 lbs.

Statistical Analysis

To keep the statistical analysis within manageable bounds, the analysis was limited to the *independent* variables:

- x_1 length of treatment (in months)
- x_2 average dosage (in cc's)
- x_3 starting weight (in pounds)
- x_4 age (in years)
- x_5 sex (coded 0 for female—1 for male)

and the *dependent* variables

y weight loss (in pounds)

(1) A preliminary investigation also showed that the correlation between age and weight loss is $r = -0.29$, which is not statistically significant at the 0.05 level.

Preliminary investigations showed that the correlation between sex and weight loss is 0.01, which is negligible. Actually, the data support the hypothesis that the treatment applies equally well to men and women.

(2) A multiple regression analysis applied to the other variables yielded the following equation:

$$y = 0.52x_1 + 5.42x_2 + 0.172x_3 - 24.34$$

and a multiple correlation coefficient of $R = 0.63$. This means that appoximately 40 per cent of differences in weight loss among patients can be explained (attributed to) the three independent variables. The other 60 per cent may be due to chance variation or other factors not taken into consideration. To illustrate the use of the regression equation, suppose that a patient whose initial weight is 200 pounds is to be given an average dosage of 2 cc for 12 months. Substitution into the equation yields:

$$y = 0.52(12) + 0.172(200) - 24.34$$
$$= 27 \text{ pounds (approximately)}$$

In other words, under the given conditions you can predict a weight loss of 27 pounds.

Summary

The author therefore feels the study shows conclusively that a relatively new application of an already existing medication (intramuscular dextro-amphetamine sulfate) in very large doses, several times a day is a good, effective and *safe* treatment for intractable obesity, and its complications. Intractable obesity is that which does not or cannot respond to oral anorexic agents or dietary management when no endocrine abnormality exists.

Many patients who cannot tolerate 15 mg of dextro-amphetamine sulfate by mouth can comfortably take 30 mg by the intramuscular route without disconcerting side effects. Doses used have been as high as 100 mg per day for extended periods. The usual method is either self-administered at home the first thing on arising or at 8:00 a.m. in the office daily for as much as several years running. The 100 mg doses were divided, 60 mg in the morning and 40 mg in the early afternoon. No marked local irritation at the site of injection was found in any patient and a male patient who has taken as high as three injections per day for three years, with an average dose of one injection per day, noted no irritation at all. The chief value was in the intractable obese patient with hypertension, cardiac decompensation, or orthopoedic problems such as repeated fractures or chronically strained, sprained ankles and osteo-arthritis of the knees. Post coronary rehabilitation, especially where aging is a complication of obesity, also was aided greatly by this method. In several isolated cases, non-specific headache was relieved and severe angina was improved.

Since a complete double-blind controlled study with an ineffective substance is not feasible, for economical reasons, in private practice, two cases were followed, who had placebos for one month.

The first was a 50-year-old male who took 2 cc of sterile water every morning for one month following a 59-pound weight loss over one year on dextro-amphetamine sulphate intramuscularly. He noticed a let-down in his energy level and his weight remained stationary.

A second case was a 53-year-old female who had lost 20 pounds in four months on dextro-amphetamine sulphate intramuscularly. When a new pharmacist mistakenly filled her prescription, which was written for the trade name 'Dextromine' (dextro-amphetamine sulphate), and gave her instead 50 per cent dextrose solution, she gained 5 pounds in the month that transpired before the error was found. She noticed a marked decrease in her feeling of well being during this period and informed me that something had surely gone wrong with the medicine.

(Note: Statistical analysis done by John Freund, Ph.D., Arizona State University.)

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Letter to a Probation Officer on a Case of Homosexuality

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The case which motivated my letter herewith transcribed was a very interesting one from many points of view and, especially, because it actualized the old question of how medical science may help the homosexual. This letter is the answer to a Probation Officer, appointed by the Court to supervise a young sexual pervert, and in it is discussed some of the medical, legal and social problems that homosexuality creates. That is why, I think, that its publication may be of some interest to my colleagues.

Dear Mr. M. . . . . .

This letter is in reference to Mr. Peter M., a previous patient in this Unit, who was released on . . . . . I have been quite concerned ever since in trying to secure the best solution to his problem, and, as I promised you in our telephone conversation, in the following I will try to discuss this case and summarize the conclusions at which I have arrived.

1) Although the diagnosis in this specific case is more an academic matter, I would like to express my opinion that Peter is not suffering from a true psychotic disorder. I know that he was diagnosed before as a schizophrenic; however, in my interviews, as well as in the observation of his behavior during his stay in the hospital, no symptoms or signs on which to base such a diagnosis were found.

I think he belongs to the group that modern psychiatry knows as sociopathic personality, sexual deviation (also called sexual perversion), in whom the only manifestations of the disorder are in the sexual sphere. The pervert suffers from an anomaly of the sexual drive and gets satisfaction either in some other activity than that of complete heterosexual intercourse, or, in some deviant activity, acts that are not accepted by our morals, customs or laws.

Peter, as the generality of homosexuals, has a tendency to be immature in his reactions, is easily depressed and discouraged, frequently frustrated, emotionally unstable, dependent and self-indulgent, and involved in love affairs with other men which end in disappointments, frustrations and suicidal thoughts. These could have the appearance of psychotic symptoms, but, altogether, do not constitute the well-defined picture that characterizes the schizophrenic.

2) The etiology of homosexuality is not yet known. There are various theories which command a varying amount of evidence in their support. Disorders of the endocrine system, hereditary predisposition, and homosexual seduction during infancy play a role in some individual cases, but it is impossible to consider all homosexuals as the product of only one factor.

Various theories of a psychogenic causation have been put forward and it is psycho-analysis which has the larger number of supporters. Psychoanalysis maintains that what we call normal sexuality in the adult is the end product of a large process that starts from the beginning of childhood. In brief, the child is born with all the germs of sex which gradually develop into what one later calls sex. During the development of the individual and due to various influences in his relationship with the objects of the environment,

mainly unconscious or conscious parental attitudes, the diverse components of the sexual drives can suffer an arrest in a period of its normal evolution or even a regression to a previous phase of its development as well as other types of disorders through psychological mechanisms (fixation, regression, identification, etc.).

In the case of Peter, we find in his background many of the factors mentioned and we can elaborate a great deal regarding the psychodynamics, the psychological forces that have been acting in this youth since his childhood and made him what he is. The problem, as I see it from a practical standpoint, is that we are dealing with a youngster, who at the present time shows all the emotional feelings of a female, even though he has the complete appearance of a male. Due to his abnormal urges he has been indulging in homosexual relations and creating a difficult problem in his community.

3) What to do with a case as this one? What are the solutions, if any, that we can offer? What are the resources available to reach those solutions?

The case of Peter M . . . creates a medical, a legal and a social problem.

First of all, I would like to point out that our morals, culture and entire life are based on the differences of sex, namely, a person is totally male or completely female. Under our standards, according to the Judeo-Christian tradition, we do not admit the existence of individuals who are in the middle of the scale of sexuality and according to the same tradition procreation is the only legitimate aim of sexual activity. Therefore, there is a tendency to condemn all forms of sexual activity not directed toward that aim. Mosaic law prescribed the death penalty for these offenses. The Medieval Church, as well as secular powers, inflicted extremely severe punishment, including death by burning or by being buried alive, for persons convicted of sodomy.

Although a more humane treatment of homosexuals began in the 19th century with the Napoleon Code, which abolished all the penalties for homosexual acts occurring between adults in private and without violence, in the statutes of almost all countries, as well as in all the states of this country, there are established punishments for these so called crimes. New Mexico Statutes contemplate this offense in Section 40-7-6, 40-7-7, 40-7-8 ruling that any person convicted of this offense "shall be imprisoned for less than one (1) year, or fined in any sum not less than $1,000, or both, in the discretion of the court".

It is not only the law which condemns sexual deviations. The attitude of the community, as you pointed out in your letter, is still one of contempt and moral condemnation. In regard to homosexuality in particular, a great deal of opprobrium still attaches to it.

"Actually," say English and Pearson in the book, *Emotional Problems of Living*, "society condemns sexually perverse behavior without knowing exactly why. People lead their sexual lives in private, and how they conduct their sexual relations need be none of any others' business. Nevertheless, society has a tendency to be upset about it, as if this type of behavior contaminated everyone in some way, and people act as though condemnation could bring about its reduction. We would like to make clear that society can never bring about the reduction of perverse sexual behavior through condemnation or through punishment of the sexually perverse individual, whether by excluding him from every day life or from society or by bringing him into court and putting him in jail. These attempts do nothing to change him; he will go on with his perverse behavior just the same; society will never understand what causes it and hence will never cure it . . . For he is driven on by strong instinctual forces of which he is not aware, forces of which doctors and scientists have to be aware if they hope to alleviate the problem. The sexually perverse person is often hated, lonely, and in serious conflict about his problem."

In the matter of sexuality we cannot be too rigid. It is an Utopia to divide mankind in absolute female and absolute male. There are degrees, a rate in scale which goes from the exclusively heterosexual individual to the exclusively homosexual, and even embryologically every man has some remnants of female and every female has some remnants of male. Recently a new important discovery shows that the cells of the body have sex, and although a person could have all the appearances of one of the sexes, the sex of his cells can be of the opposite one, a discovery that perhaps will provide a biological reason for the perplexing sexual behavior of the homosexual.

We can conclude, in the light of our present knowledge in this matter, that our laws and the community attitudes in this respect are not only unscientific but unjust. They are based on ignorance and strongly determined by unconscious as well as conscious fears rather than on an objective evaluation of facts, and the legal implications of homosexuality and other sexual deviations are still to be put on a scientific or humane basis.

a) From the psychiatric and medical point of view, the first question that arises in cases as the one we are discussing is whether or not a person with the characteristics of Peter, I repeat, mentally female and physically male, could be converted into a totally male or totally female individual.

Peter came to this hospital with the idea that an operation could be performed to make him apparently, at least, more female. In other words, he completely refused the idea to become a male; even more, he was disgusted, disappointed because his physical appearance did not fit with his female mind and he thought that medical science could convert him into what he has been longing to be.

Indeed, that kind of operation has been performed in some exceptional cases. In this country the case of Chris Jorgenson is fairly well known. He was a normal man in body and general appearance, and served for a time in the United States Army, although his mind was totally female. He went to Denmark where his testicles were removed and came back more satisfied with his appearance, enjoying life more than before when he was obliged to play the role of a man.

Can we advise such an operation in the case of Peter M . . . ? There are many factors to be considered. In fact, could we legally sanction such an operation? Should a surgeon agree to perform it? Is it justified from the religious point of view to try to transform what God decided? In the event that the operation is performed, should he be considered as a man or as a woman in spite of the fact that he will be lacking the male sexual characteristics as well as those of a female.

I feel that with all these drawbacks, we can hardly advise such a porcedure and, practically, we rule it out as a prospective solution of this problem.

b) Sex hormone therapy could be another possibility for treatment of this case, in order to accentuate the masculine characteristics or to the contrary to increase his female tendencies. Whatever may be the goal, it is recognized that this kind of treatment is unable to secure a true change of sex and in the best of cases its effects would be only a slight accentuation, if any, of the patient's urges in one of the two sexual behavior patterns. Therefore, we also rule out this procedure.

c) Some authors suggest electroshock treatment in cases such as this one. This kind of treatment also deserves a brief discussion. First of all, we do not know its mechanism of action and aside from specific indications, it is not justifiable to expose a person to its risks or hazards. The amnesia that electroshock produces, that could be the basis for its use, is temporary, and when it is over, the abnormal urges or tendencies return to the surface with their previous energy. That is why I deem it useless in Peter's case. The same could be said in regard to hypnosis, as it is recognized that the results achieved have been very limited and short lived.

d) Finally, psychotherapy and particularly psychoanalysis is deemed the treatment of choice for homosexuals, the aim being not to try to change them, which all the authors agree is extremely difficult and perhaps impossible, but to adjust the patient to his inversion. However, the pervert derives conscious gratification from his sexual deviation, and it is exceptional that he seeks treatment on account of his perversion unless he is forced to do so because of external pressures, his family, the legal authorities or other agencies of the community.

That is the reason why psychotherapy, although considered the treatment of choice, has failed in most of the instances, even in the hands of so famous a therapist as Freud.

"We have to realize," say the authors previously mentioned, "that homosexuals are seriously maladjusted persons whom we cannot always cure but whom we can help by seeking to alleviate the emotional tensions in the situation. The homosexual problem is a large one; we can cure homosexuality no more easily than we can cure severe neurosis. Because of certain sources acting over a period of many years, the homosexual has achieved an end point that is very difficult to change. He complains that society does not give approval to homosexual activity, yet at the same time he is

quite satisfied with his means of gratification. When heterosexuality is offered to him — no matter how attractive it looks to us — he cannot adopt it. Furthermore, homosexuals do not come to physicians very often when in trouble because they cannot believe they will be understood."

Even when the removal of a person's perverse symptom proves impossible, as I think is the case of our patient, psychotherapy still can help him to reach a better social adjustment. We must accept the fact that there have been so many conditions inclining him in the direction of homosexuality that with our present resources we are unable to effect a cure or change.

"There are times," express English and Pearson, "when we have to be 'just practical' and accept the fact of homosexuality in certain people and try to help them live as happily and efficiently as possible. That is our task as doctors. It is tremendously important that as doctors we know what human beings are like, that we accept them as we find them, that we use a certain good judgment in trying to help them get along by making the best of what we find in them. . . . On the other hand, as long as they can function fairly well as homosexuals, they can at least get along and contribute as social beings. This may not be an 'idealistic' viewpoint but at least it is a workable and practical one."

As a summary of what we can expect of psychotherapy in the case in study, I cannot avoid the temptation to transcribe a letter by Sigmund Freud to an American woman who appealed to him for advice about her homosexual son Jones E. "The Life and Work of Sigmund Freud," the last phase 1919-1939, Vol. 3, Basic, New York, 1957).

Dear Mrs. .....

I gather from your letter that your son is a homosexual. I am most impressed by the fact that you do not mention this term yourself in your information about him. May I question you, why you avoid it? Homosexuality is assuredly no advantage, but it nothing to be ashamed of, no vice, no degradation, it cannot be classified as an illness; we consider it to be a variation of the sexual function produced by a certain arrest of sexual development. Many highly respectable individuals of ancient and modern times have been homosexuals, several of the greatest men among them (Plato, Michelangelo, Leonardo da Vinci, etc.). It is a great injustice to persecute homosexuality as a crime, and cruelty too. If you do not believe me, read the books of Havelock Ellis.

By asking me if I can help, you mean, I suppose, if I can abolish homosexuality and make normal heterosexuality take its place. The answer is, in a general way, we cannot promise to achieve it. In a certain number of cases we succeed in developing the blighted germs of heterosexual tendencies which are present in every homosexual, in the majority of cases it is not more possible. It is a question of the quality and the age of the individual. The result of treatment cannot be predicted. What analysis can do for your son runs in a different line. If he is unhappy, neurotic, torn by conflicts, inhibited in his social life, analysis may bring him harmony, peace of mind, full efficiency whether he remains a homosexual or gets changed. If you make up your mind he should have analysis with me!! I don't expect you will!! He has to come over to Vienna. I have no intention of leaving here. However, don't neglect to give me your answer.

Sincerely yours with kind wishes,

Freud."

e) I have also been trying to explore any other possibility of helping this youth. In our telephone conversation I said I knew of a special institution in California that takes care of sexual perverts, but lately I learned that actually it is a hospital for psychotics, in whom the sexual deviation is one of the symptoms of the disease and not a manifestation of a pure sexual perversion. On the other hand, there is no special treatment that could be considered a hope for our patient, and admissions are limited to residents of that state.

Labortherapy may also be, as you very well pointed out, good. With this in mind I contacted Mr. D. . . . of the Vocational Rehabilitation Department asking him about the possibility of placing Peter in a vocational program as a beautician, since he expressed interest in that type of work and since it will fit his aptitudes and general make-up. He told me that Peter could have good chances for such a program, provided that he wear clothes according to his sex, which, as you know, the patient refuses to do.

f) I left as the last point to comment on the possibility of hospitalizing this patient in our institution. From the community's standpoint apparently this is the best solution. With such a decision the problem is removed from the community, which rejects him for reasons I tried to summarize at the beginning of this letter. If we consider the implications of his hospitalization from the point of view of the patient himself, we arrive at another conclusion. As a matter of fact, what could be the benefit that he can obtain from being hospitalized? If we start with the premise that Peter is not psychotic, he is not a fit person to be hospitalized. To oblige him to live and associate daily with mentally sick persons will be more detrimental to his already seriously disturbed personality than to stay in the community. Furthermore, due to his sexual deviation, he is prone to be involved in love affairs with other patients, who for reason of their mental status, lack of judgment and insight, are easy candidates for his advances with the natural harm to them, as well as to the order and discipline of the institution.

In any event, if we admit him here, in spite of the above-mentioned drawbacks, how long should he be kept here? Weeks, months or years?

As I said before, I do not think that we can change Peter's condition. No matter how long he would be hospitalized, as soon as he were released he would return to his previous homosexual experiences, pushed by the force of his deviated urges.

For the above-mentioned considerations, hospitalization in this case is not advisable.

Having discussed very briefly the possibilities of treatment and the prospective result of each of them in this specific case, I will now try to summarize my recommendations:

1) Take an understanding attitude toward his sexual behavior by explaining to his family, his relatives and members of the community that Peter M. should be accepted the way that he is.

2) Alleviate his emotional tensions, his frustrations, anxieties and periods of depression. In this sense, psychotherapy, adjusting him to his inversion, is the type of therapy recommended, if financially feasible. Some psychopharmacologic agents could also help him in achieving this end.

3) Punishment is by no means indicated. The best thing one can do is treat him as politely as one would anyone else. He, on his part, of course, should be expected to abide by the ordinary rules of decency such as applied to relationship between men and women, namely, he should not seduce others nor force himself on people who are not interested in his company. He should not flaunt his desires in public by dressing in clothes of the opposite sex or otherwise and he should not embarrass those around him by making love or talking about it in public.

   If he behaves himself and controls himself as discreetly as people with heterosexual desires are expected to do, his private life should be of no more concern to anyone else than should a normal person's.

   Putting him in jail or in a hospital results only in providing him and the other inmates or patients with added opportunities for abnormal sexual activity.

4) Due to the tendencies of being immature in his reactions, easily depressed, discouraged and frequently frustrated, he could be a suicidal risk; therefore, close supervision by the Probation Officer is in order.

5) The tentative idea of placing him in Vocational Program for the purpose of training him as a beautician should be encouraged, if he would agree to dress as a man during the training period.

6) It is also felt that a priest could help by providing him with religious support.

I regret the delay in answering your letter and especially the fact that an effective program, that could solve this difficult case, is not available. I feel that we can do no more than what I expressed to you in this long letter.

I am very interested in this case and I would greatly appreciate progress reports on Peter on how he is getting along. If I can be of further assistance, please feel free to write to me at anytime.

Sincerely yours,
(Sgd.) R.M.B.

Letter to the Editor

Let us accept the Challenge!

The Doctors were not defeated in the past Election. The Government of the United States of America is not an enemy of the Medical Profession.

Let us accept the challenge and find — as always — our proper place in this new and ever more complex world in which we live. Let us think first — as always — of our PATIENTS. Let us resolve — as always — that EVERY patient in his struggle to live gets the very best of medical care when and where he needs it.

The Doctor fits in as a NECESSITY in any society. He can survive no matter what. He survived in the Great Depression when his fee was often a sack of potatoes or a quarter of beef, and he has fared well since the Depression and shared in the general prosperity. He did his bit in the Wars.

The Doctor as an individual enjoys the highest esteem of people. He earned this esteem by kindness, by keenness of mind and by faithful execution of duty. He is among the first to manifest his principles that all people should have the benefit of his knowledge regardless of economic condition.

Most of the new changes will benefit the practice of Medicine. The Doctors should take their position with leaders and make sure that those changes actually benefit those who need medical care. The Doctor-Patient relationship is sacred and should be preserved.

W. E. Lockhart, M.D.
Alpine, Texas

Announce Speaker For S.W. Medical Ass'n. Meet

Dr. Donald G. Mulder, Associate Professor of Surgery at the University of California at Los Angeles School of Medicine, will be a member of the Faculty at the 47th annual meeting of the Southwestern Medical Association in El Paso, November 4-6, 1965, Dr. A. Robert Nering of El Paso, Program Chairman, has announced.

The program will be presented by the University of California at Los Angeles School of Medicine. General subject for the session will be Chest Diseases.

Dr. Mulder has been a member of the fulltime staff of UCLA since 1957 and he is primarily interested in thoracic surgery, particularly cardiac procedures. He received his M.D. from Johns Hopkins University and took his postgraduate surgical training at the Johns Hopkins Hospital and at the UCLA Medical Center.



Dr. Mulder

Dr. Gordon Black of El Paso is General Chairman for the meeting. Dr. Clement C. Boehler, El Paso, is President of the Association.

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Coming Meetings

New Mexico-El Paso Chapter of the American College of Surgeons, Ruidoso, N. M., September 11-12, 1965. Headquarters: Chaparral Motel.

62nd Annual Meeting of the Western Association of Railway Surgeons, Alameda Hotel, Avenida Juarez 50, Mexico City, October 27-30, 1965.

15th Annual Meeting of the Southwest Obstetrical and Gynecological Society, Arizona Inn, Tucson, Oct. 28-30, 1965.

47th Annual Meeting, Southwestern Medical Association, Sheraton Motor Inn, El Paso, November 4-6, 1965.

10th Annual Clinical Conference, University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, Nov. 5-6, 1965.

17th Annual Western Institute on Epilepsy, Kona Kai Club, Shelter Island, San Diego, Calif., Nov. 11-13, 1965.

Inaugural Scientific Program of the new Ancker Hospital, Saint Paul, Minn., Nov. 11-13, 1965.

10th Annual Meeting of the Medical Society of the United States and Mexico, Hermosillo, Mexico, Nov. 18-20, 1965.

99th Annual Session of the Texas Medical Association, Austin, April 14-17, 1966.

10th Congress of the Pan-Pacific Surgical Association, Part I: Honolulu, Sept. 20-28, 1966. Second Mobile Educational Seminar, Part II: Japan and Hong Kong, Sept. 28-Oct. 10, 1966. Part III: Japan, Hong Kong, The Philippines, Thailand, India, Singapore, Australia and New Zealand, Sept. 28-Nov. 1, 1966.

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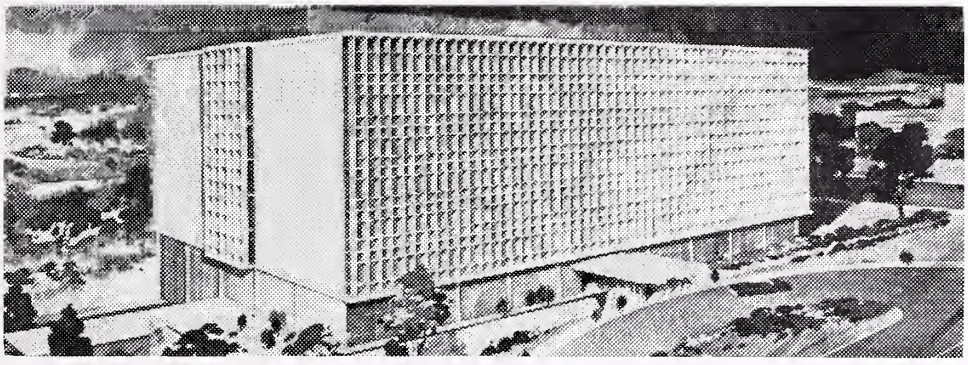
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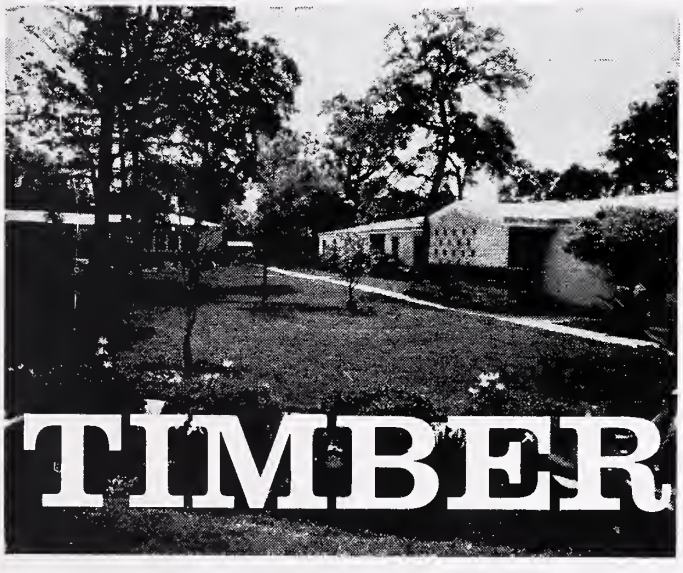
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Side effects: Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

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Your attention is invited to Page 285 for details on the Southwestern Medical Association Low Cost Group Life Insurance and Other Insurance Programs for you, your wife and family.

VOL. 46, NO. 9

September, 1965

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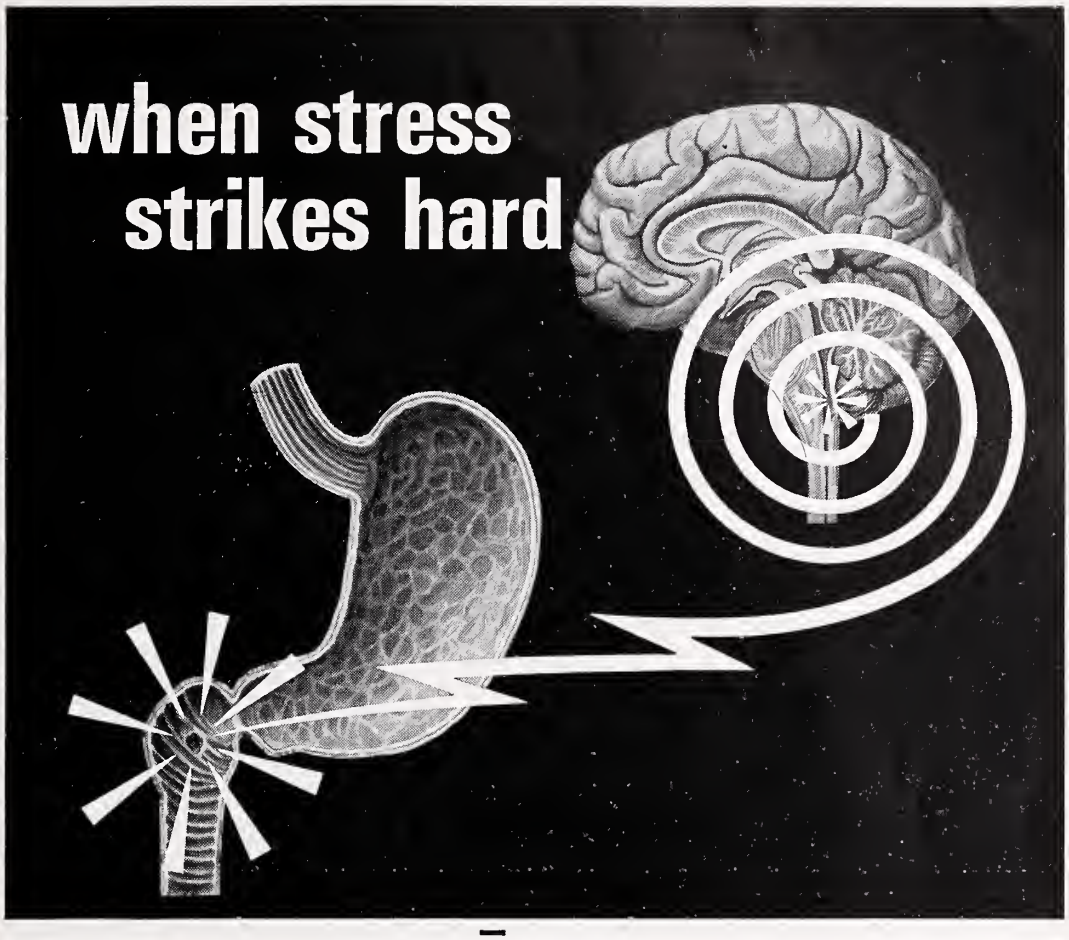
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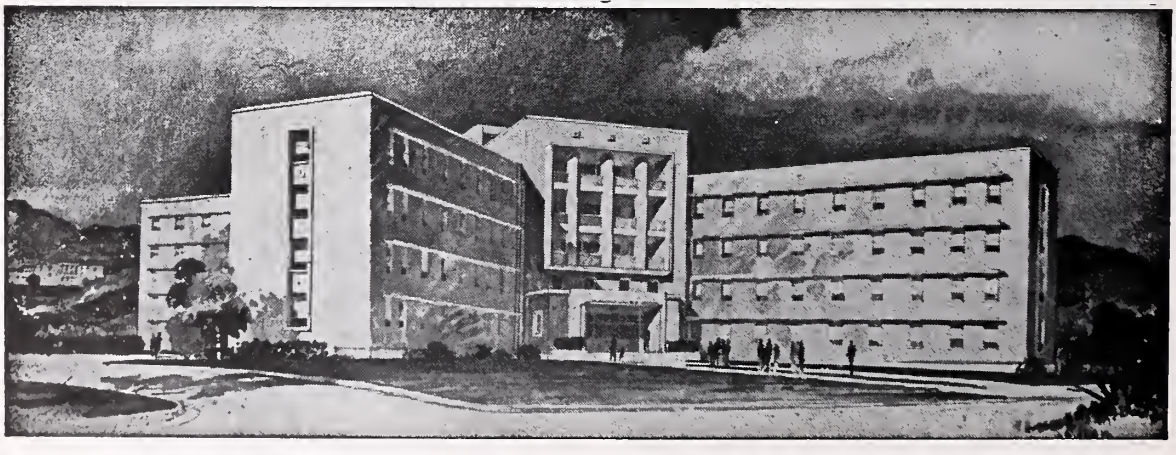
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The teen-ager is not blessed with an inherent or acquired capacity to store essential amino acids against the time when they may be in short supply. Animal studies indicate that a substandard intake of any one essential amino acid may serve as a limiting factor in the utilization of all. To assure maximal growth and efficient tissue production, the teen-age daily diet should contain adequate amounts of all essential amino acids along with sufficient calories and other essential nutrients. The optimal daily protein requirement during adolescence has been estimated at 15 percent of the caloric needs of the individual.

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Precautions: *Meprobamate*—Careful supervision of dose and amounts prescribed is advised. Consider possibility of dependence, particularly in patients with history of drug or alcohol addiction; withdraw gradually after use for weeks or months at excessive dosage. Abrupt withdrawal may precipitate recurrence of pre-existing symptoms, or withdrawal reactions including, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, the dose should be reduced and operation of motor vehicles or machinery or other activity requiring alertness should be avoided if these symptoms are present. Effects of excessive alcohol may possibly be increased by meprobamate. Grand mal seizures may be precipitated in persons suffering from both grand and petit mal. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side effects: Side effects associated with recommended doses of 'Deprol' have been infrequent and usually easily controlled. These have included drowsiness and occasional dizziness, headache, infrequent skin rash, dryness of mouth, gastrointestinal symptoms, paresthesias, rare instances of syncope, and one case each of severe nervousness, loss of power of concentration, and withdrawal reaction (status epilepticus) after sudden discontinuation of excessive dosage.

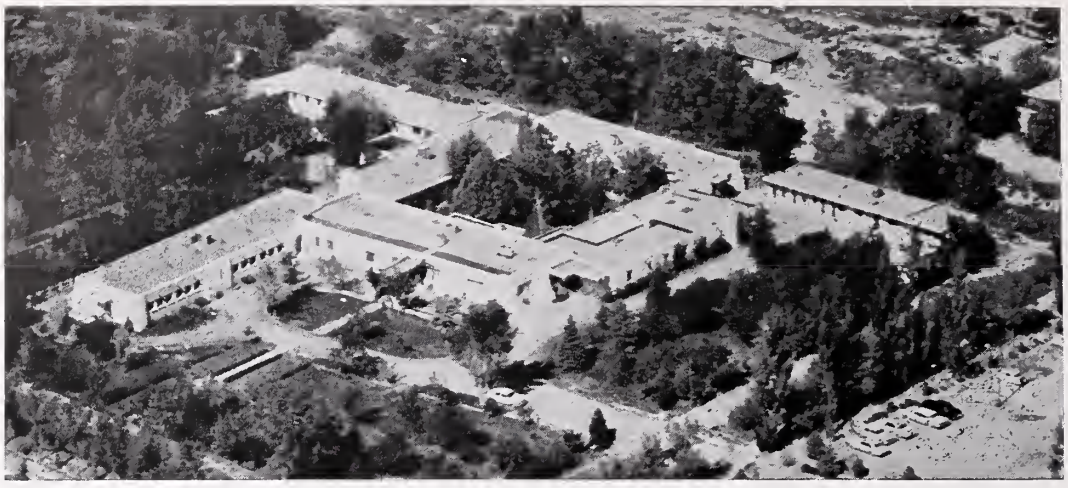
Benactyzine hydrochloride—Benactyzine hydrochloride, particularly in high dosage, may produce dizziness, thought-blocking, a sense of depersonalization, aggravation of anxiety or disturbance of sleep patterns, and a subjective feeling of muscle relaxation, as well as anticholinergic effects such as blurred vision, dryness of mouth, or failure of visual accommodation. Other reported side effects have included gastric distress, allergic response, ataxia, and euphoria.

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Dosage: Usual starting dose, one tablet three or four times daily. May be increased gradually to six tablets daily and gradually reduced to maintenance levels upon establishment of relief. Doses above six tablets daily are not recommended even though higher doses have been used by some clinicians to control depression and in chronic psychotic patients.

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95th Annual Session, Colorado Medical Society, Broadmoor Hotel, Colorado Springs, Colo., Sept. 19-22, 1965.

13th Annual Meeting, Arizona Academy of General Practice, Del Webb's Towne House, Phoenix, Oct. 7-9, 1965.

62nd Annual Meeting of the Western Association of Railway Surgeons, Alameda Hotel, Avenida Juarez 50, Mexico City, October 27-30, 1965.

15th Annual Meeting of the Southwest Obstetrical and Gynecological Society, Arizona Inn, Tucson, Oct. 28-30, 1965.

47th Annual Meeting, Southwestern Medical Association, Sheraton Motor Inn, El Paso, November 4-6, 1965.

Interim House of Delegates Meeting of New Mexico Medical Society, Holiday Inn, Clovis, New Mexico, Nov. 4-5, 1965.

10th Annual Clinical Conference, University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, Nov. 5-6, 1965.

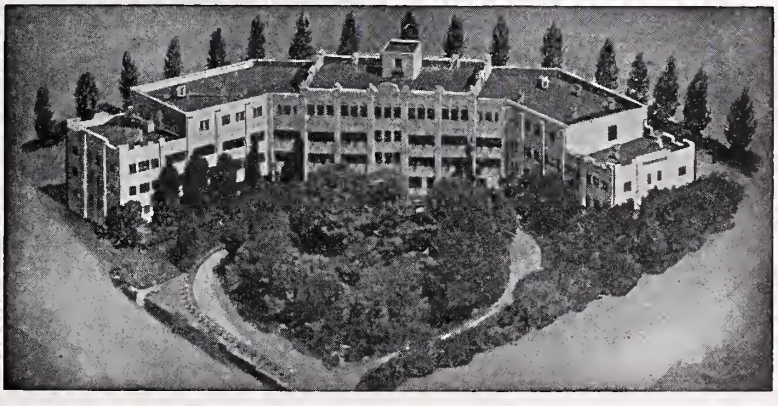
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Announce Program for Southwestern Medical Ass'n. Meeting El Paso, Nov. 4-6

The 47th annual meeting of the Southwestern Medical Association will be held in El Paso, November 4-6, 1965, with members of the Faculty at the meeting to be provided by the University of California at Los Angeles School of Medicine. General subject for the meeting will be Chest Diseases.

Members of the Faculty are as follows:

Arthur A. Clinco, M.D., Associate Clinical Professor of Psychiatry.

Donald G. Mulder, M.D., Associate Professor of Surgery.

John F. Murray, M.D., Associate Professor of Medicine and Physiology.

Justin J. Stein, M.D., Professor of Radiology and Chief, Radiotherapy Division.

The three-day program, with scientific sessions in the morning only, will be presented under the direction of Dr. Charles R. Smart, Assistant Professor of Surgery in Residence and Assistant Director of Postgraduate Medical Education. The program has been approved by the Texas Academy of General Practice.

Headquarters for the meeting, which will be open to all physicians, will be at the Sheraton-El Paso Motor Inn. Registration is $15.

Dr. Clinco is a graduate of the Columbia Psychoanalytic Institute, a Diplomate in Psychiatry and Neurology, a Fellow of the American Psychiatric Association and the Academy of Psychoanalysis, a Charter Member of the American Academy of Neurology, and a Past President of the Southern California Psychoanalytic Society.

Dr. Mulder received his M.D. from Johns Hopkins University and took his postgraduate surgical training at the Johns Hopkins Hospital and at the UCLA Medical Center. He has been a member of the fulltime staff of UCLA since 1957 and he is primarily interested in thoracic surgery, particularly cardiac procedures.

Dr. Murray received his M.D. from the Stanford University School of Medicine and is a Diplomate of the American Board of Internal Medicine and its Subspecialty Board of Pulmonary Diseases. He was a Research Fellow of the American College of Physicians, Postgraduate Medical School of London, 1956-1957. Last year he was President of the Western Section of the American Federation for Clinical Research and also was Visiting Scientist of the Cardiovascular Research Institute at the University of California at San Francisco.

A graduate of Baylor University College of Medicine, Dr. Stein is a Diplomate of the American Board of Radiology and is a Fellow of the American College of Surgeons, the American College of Radiology and the International College of Surgeons. He is President of the American Radium Society, a member and Past President of the California State Board of Medical Examiners, and is Director of the California Institute for Cancer Research.

Officers of the Southwestern Medical Association are Dr. Clement C. Boehler, El Paso, President; Dr. Frank A. Rowe, Albuquerque, President-Elect; Dr. Zigmund W. Kosicki, Santa Fe, Vice-President, and Dr. Sol Heinemann, El Paso, Secretary-Treasurer. Dr. Gordon Black of El Paso is General Chairman and Dr. A. Robert Nering, El Paso, is Program Chairman.

The complete program is as follows:



Dr. Mulder

November 4, 1965

9:00-9:30	"An Evaluation of Cardio-pulmonary Symptoms", John F. Murray, M.D.
9:30-10:00	"Results of Surgical Treatment of Carcinoma of the Lung", Donald G. Mulder, M.D.
10:00-10:30	Coffee Break
10:30-11:00	"Results of Irradiation Therapy in Carcinoma of the Lung", Justin J. Stein, M.D.
11:00-12:00	Panel on above topics: Moderator: John F. Murray, M.D.

November 5, 1965

9:00-9:30	"The Psychodynamics of Respiratory Behavior", Arthur A. Clinco, M.D.
9:30-10:00	"Management of Emphysema and Bronchiectasis", John F. Murray, M.D.
10:00-10:30	"Management of Mediastinal Tumors", Donald G. Mulder, M.D.
10:30-10:45	Coffee Break
10:45-11:45	Panel on above topics Moderator: Justin J. Stein, M.D.

November 6, 1965

9:00-9:30	"Management of Inflammatory Pulmonary Disease", John F. Murray, M.D.
9:30-10:00	"Current Status of the Surgical Treatment of Congenital and Acquired Heart Disease", Donald G. Mulder, M.D.
10:00-10:30	"Modern Management of the Lymphomas", Justin J. Stein, M.D.
10:30-10:45	Coffee Break
10:45-11:15	"Psychiatry for the Non-Psychiatrist", Arthur A. Clinco, M.D.
11:15-12:00	Panel Discussion Moderator: John F. Murray, M.D.



Dr. Clinco



Dr. Murray



Dr. Stein

APHORISMS and MEMORABILIA*

(Continued)

ANDREW M. BABEY, M.D., *Las Cruces, New Mexico*

31. Only a very small portion of trichinosis cases are clinically overt. It is said that the United States has the highest trichinosis rate in the world (16%). Government inspection and marking of meat has probably done more harm than good in decreasing human trichinosis, because it has engendered false confidence. To get the stamp, a batch of meat merely has to show in one tiny sample fragment no larvae by trichinopress examination. In any clinical problem, beware of the muscle biopsy report of trichinosis: it proves trichinosis but that is not necessarily the answer to the clinical problem. Keep in mind that, if biopsied in the right place, 16 per cent of your patients will show trichinosis. (pg. 64)

32. Suspect strongyloidiasis in anyone with eosinophilia and the x-ray diagnosis of duodenitis. It can cause some of the highest eosinophilias found in your practice. (pg. 65)

33. It is said that lacerations of the spleen never heal—no instance of healed laceration has been reported at autopsy. (pg. 68)

34. Rupture of the spleen does not cause peritonitis, in contrast to rupture of the liver and pancreas. Different people react differently to blood in the peritoneal cavity—often there appears to be little evidence of peritoneal irritation. (pg. 68)

35. In one to seven cases of traumatic rupture there is a dangerously misleading latent interval between the trauma and the rupture (period of Baudet). This may last a few hours or, rarely, as long as a few months (usually about a week). Don't fail to respect a history of trauma in the recent past. Sudden death from delayed splenic rupture, occuring after an accident victim has been in bed a couple of weeks, too often is assumed to be due to pulmonary embolism. (pg. 69)

36. In feeling a spleen, pay special attention to the configuration of the edge. The experts are continually misinterpreting sharp-edged enlarged left liver lobes for spleens. (pg. 70)

37. Only about 10-15 per cent of patients with Weil's disease are sick enough to show both jaundice and oliguria. (pg. 72)

38. It has been said, "Suspect Weil's disease in any febrile patient with a striking degree of ocular injection." (pg. 72)

39. On occasion, the cirrhotic will have sudden attacks of acute abdominal pain, simulating an acute surgical abdomen. Clinical picture often encourages laparotomy, but no explanation for the pain can be found. (pg. 72)

40. Only about one per cent of spiders in cirrhosis and in pregnancy occur below the level of the umbilicus. (pg. 74)

41. But spiders may appear and disappear, apparently spontaneously, almost overnight. Most are static over fairly long periods. (pg. 74)

42. X-ray examination is able to demonstrate esophageal varices in only 24 per cent of patients who can be shown by esophagoscopy to have them (Am. J. Roentgenol., *73,* 387, 1955). (pg. 80)

43. Discovery that esophageal varices may be encountered as soon as seven days after the onset of acute hepatitis is one more bit of evidence that the native esophagogastric venous connections may quickly be mobilized to shunt off blood from a hypertensive portal bed. (pg. 81)

44. In cases of "post-cholecystectomy syndrome" it often proves worthwhile to obtain a cholecystogram. Although reformed gallbladder is not common, its discovery often solves a difficult problem (Am. J. Digest. Dis., *19,* 298, 1952). (pg. 83)

*From: "Pearls in Gastroenterology" by Colonel Eddy Palmer, M.C., USA, Washington, D.C. 1955. Reprinted with permission of Colonel Palmer.

The Management of Chronic Obstructive Bronchopulmonary Disease

Giles F. Filley, M.D., *Webb-Waring Institute for Medical Research, Denver*

Since destroyed lung tissue cannot be regenerated or replaced (yet), emphysema remains an incurable disease. Chronic bronchitis without emphysema responds to modern treatment and even when both diseases are present respiratory disability can often be substantially reduced. Whether or not significant prolongation of useful life is achieved as a **direct** result of therapy has not been established. Nevertheless much can be done to relieve the distress of almost all patients with these discouraging diseases.

I The Early Case

The aim of therapy should be to prevent progression of lung destruction but the difficulties are great. Even if the future victim of emphysema could be recognized early, it is not known if irreversible emphysema can be prevented. We have evidence that patients who stop smoking live longer than those who continue to smoke (1). This may only mean that only those patients stop smoking who think they have a chance of getting better — i.e. the "earlier cases". We just don't know. Stopping smoking greatly reduced cough in our patients. If we knew that emphysema was caused by the trauma of coughing or the retention of purulent secretions we could say we "prevented" emphysematous change. Unfortunately the cause of emphysema is unknown. Nevertheless, I believe that the most important thing we can do for the early case is to persuade him to give up cigarettes. Until more evidence is available this attitude will be based as much on faith as on facts.

Winter-time infections manifest by a change in sputum from clear to purulent must be treated promptly. Tetracycline and Ampicillin—one or the other—are the drugs of choice. Bronchodilators (oral, vaporized, rectal or intravenous) should be prescribed especially if significant wheeze on expiration is noticed. The drug and its method of administration should be chosen both on the basis of the patient's subjective response and on the results of spirometric testing. In the case of steroids, (? bronchodilating, anti-inflammatory, immuno-suppressive agents,) only the results of spirometric testing are trustworthy. Postural drainage and breathing exercises may be helpful in selected cases. Expectorants are usually useless, but at least rarely harmful. Intermittent positive pressure therapy **per se** is seldom advisable in the non-hospital case.

II The Severe Out-Patient (Respiratory Insufficiency)

Here the aim is not to prevent disease but to prevent the need for hospitalization. This can be accomplished by intensifying the above measures and by adding one or more of the following based on laboratory evaluation of ventilatory, cardiovascular, blood gas and electrolyte status.

1. If hematocrit is in excess of 60 per cent, phlebotomy should probably be performed.

2. If arterial P_{CO_2} is above 50 mm Hg, intensification of measures to increase alveolar ventilation should be undertaken. Often the chief problem is inability to raise secretions. Nebulized water inhalation, acetyl cysteine (by trans-tracheal instillation), nebulized heated hypertonic saline inhala-

1. Mitchell, R. S., Vincent, T. N., and Filley, G. F., "Cigarette Smoking and Chronic Bronchitis and Emphysema", JAMA, *188*, 12, April 6, 1964.

tions and special physical therapy to aid the patient in cleansing his airways may be very helpful. An occasional patient, especially if debilitated, may benefit considerably from assisted ventilation even though the effect of lowering the Pco_2 is only transient. Others may be aided by IPPB in their efforts to raise secretions. Respiratory stimulants are seldom helpful.

3. If arterial saturation is below 85 per cent (PO_2 below 50) oxygen therapy on an out-patient basis should be considered. This form of treatment must be highly individualized—from using 40 per cent O_2 to actuate the IPPB device (to provide a temporary "lift") to nearly continuous O_2 at 2-3 L/min by nasal cannula. The risk of CO_2 narcosis is real; hence, long term O_2 therapy is best started in the hospital. Oxygen is a potent drug and must be used with respect. Oxygen dependence (? addiction) has never been clearly defined but if O_2 is not really needed, the economic waste of supplying it can be enormous.

4. Congestive right heart failure often responds to the lowering of pulmonary artery pressure induced by the above measures; digitalis and diuretics will be needed only in those cases not so responding. The serum potassium level should be checked.

III The Hospitalized Patient (Respiratory Failure)

Respiratory failure has occurred when the respiratory system fails to ventilate the blood, i.e. when the arterial Pco_2 is above 50 and/or the arterial PO_2 is below 50 mm Hg. The diagnosis of respiratory failure is, of course, ordinarily made on clinical grounds: increasing dyspnea, cyanosis, signs of severe airway obstruction and cerebral irritation or depression often accompanied by evidence of acute purulent bronchitis. Unlike hypoxia there are, unfortunately, no pathognomonic signs of CO_2 retention: unexplained drowsiness is perhaps the least unreliable. Tables 1 and 2 summarize the manifestations of respiratory failure.

The circumstances in which respiratory failure occurs are listed in Table 3. It is true that patients without obstructive lung disease may develop respiratory failure, but by far the most common setting is the chronic bronchitis-emphysema group of diseases. Table 4 lists the numerous complications which pulmonary patients in failure are subject to. In Table 5 a rational approach to management is outlined.

Very intensive care is needed to combat respiratory failure in obstructed patients. In most centers more than 50 per cent of patients admitted with chronic obstructive bronchopulmonary disease in respiratory failure die in the hospital. There is every likelihood that with more intelligent use of the laboratory (for **frequent** monitoring of blood gases and electrolytes) and of mechanical devices and other means of temporarily substituting for patients' muscles and regulatory controls, this high mortality will be improved.

Of those patients with advanced emphysema who survive, few are as well as before they entered and most are worse. The patient with chronic bronchitis, secondary polycythemia and heart failure will respond to therapy better than the cachectic, dyspneic and hyperventilating case. Because improvement is unpredictable and sometimes dramatic we treat all comers as vigorously and hopefully as possible until we learn how better to determine long term prognosis.

Table 1

Clinical Manifestations of Respiratory Failure

Hypoxia	CO_2 Retention
Cyanosis	.....
Tachycardia	Tachycardia
Impaired Motor Function	Drowsiness
Confusion	Irritability
Unconsciousness	Coma

Table 5

Rational Approach to Respiratory Failure

1) Infection	Antibiotics
2) Secretions	Moisture, Remove
3) Hypoxia	Oxygen
4) "Bronchospasm"	Bronchodilators
5) CO_2 Retention	Assisted Ventilation
6) Thoracic Exhaustion	Assisted Ventilation
7) Heart Failure	The above, plus digitalis and diuretics

Table 2

Laboratory Manifestations of Respiratory Failure

Measurement (arterial blood)	Instruments
↓ Oxygen saturation, So_2	Hemoreflector, Oximeter Van Slyke, Hgb. diss. curve + Po_2
↓ Oxygen tension, Po_2	O_2 Electrode, Riley Bubble, Hgb, diss. curve + Sa_{02}
↑ CO_2 tension, Pco_2	Astrup, Electrode, Henderson-Hasselbalch Equation, Campbell Re-breathing, Riley Bubble

Table 3

Respiratory Failure Most Common In:

I Acute Infection Superimposed On Chronic Obstructive Bronchopulmonary Disease

Acute Bronchitis Acute Broncholitis Broncho-pneumonia	Complicating	Chronic Bronchitis Emphysema Asthma

II Chest Injury

Pneumothorax Intra Thoracic Hemorrhage Splinting From Pain	Causing	Atelectasis Retained Secretions Asphyxia

III Post Major Thoracic or Abdominal Surgery—Especially If

Pleural Complications Peritonitis Unrecognized Bronchitis	Causing	Increased demands for ventilation and reduced ventilatory capacity

IV Miscellaneous

Pulmonary "Fibrosis"	
Neuromuscluar Disorders	e.g. Guillain-Barre Syndrome

Table 4

Complications of Respiratory Failure

I Cardiovascular and Renal

a) Hypotension
b) Hypertension
c) Cardiac Arrythmias (sensitivity to digitalis)
d) Congestive Failure
e) Uremia

II GI

a) Gastric Distention (air swallowing or iatrogenic)
b) Ileus (with low potassium)
c) Acute Pancreatitis
d) Ulcer with Bleeding or Obstruction

III Electrolytes

a) Any combination of acid-base disturbances may occur
b) Alkalemia (↑pH) often occurs with assisted ventilation
c) Hypokalemia, Hypochloremia

IV CNS

a) Confusion, Stupor, Convulsions

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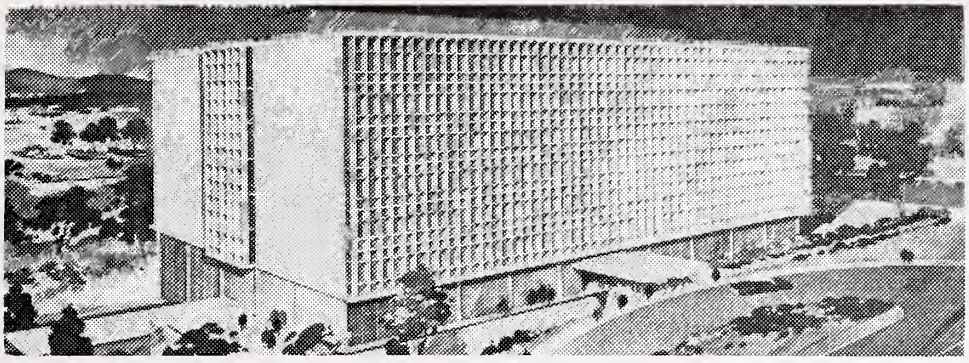
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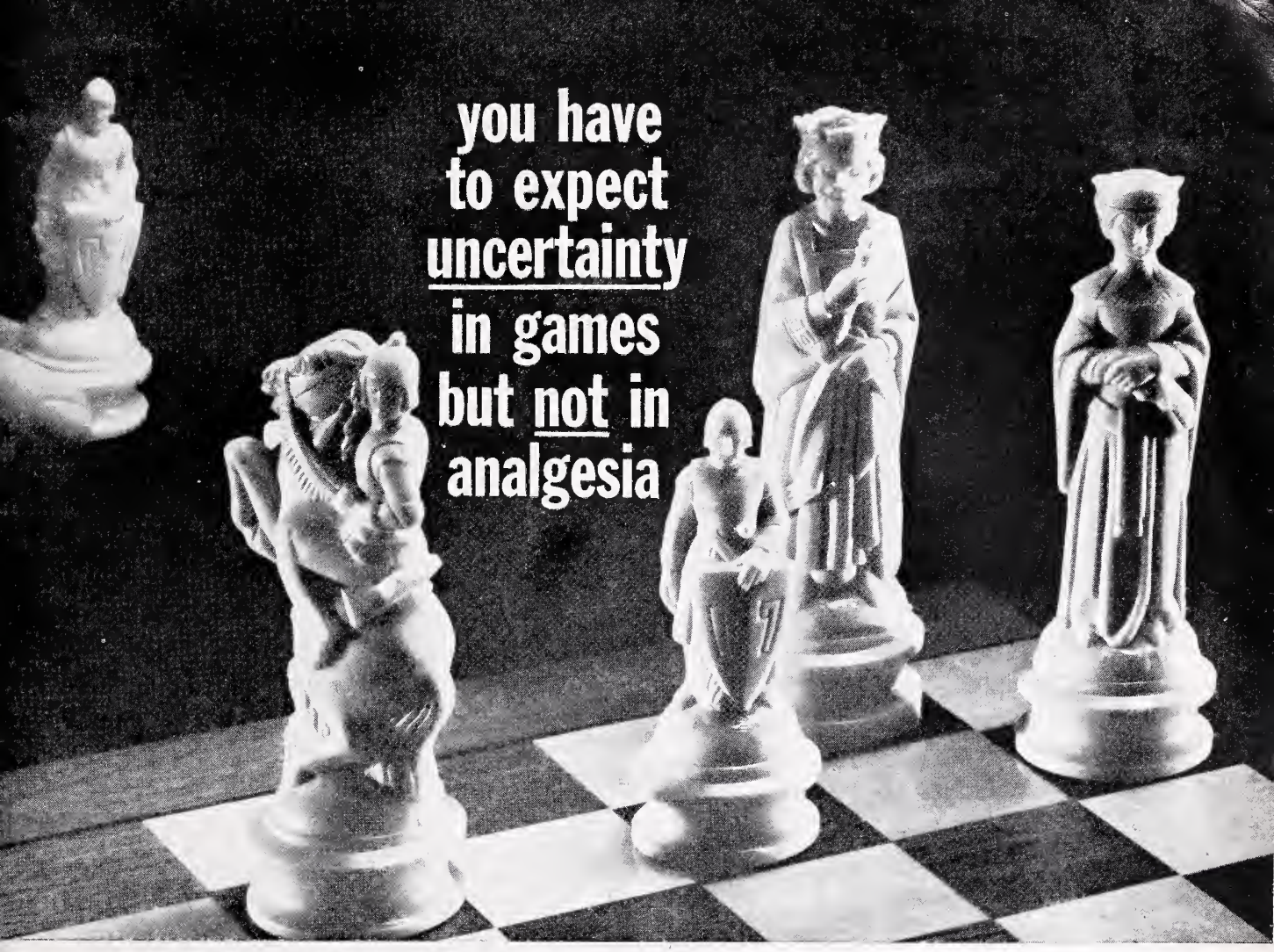
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Precautions: Careful supervision of dose and amounts prescribed is advised. Consider possibility of dependence, particularly in patients with history of drug or alcohol addiction; withdraw gradually after use for weeks or months at excessive dosage. Abrupt withdrawal may precipitate recurrence of preexisting symptoms, or withdrawal reactions including, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, the dose should be reduced and operation of motor vehicles or machinery or other activity requiring alertness should be avoided if these symptoms are present. Effects of excessive alcohol may possibly be increased by meprobamate. Grand mal seizures may be precipitated in persons suffering from both grand and petit mal. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side effects: Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

Usual adult dosage: One or two 400 mg. tablets three times daily. Doses above 2400 mg. daily are not recommended.

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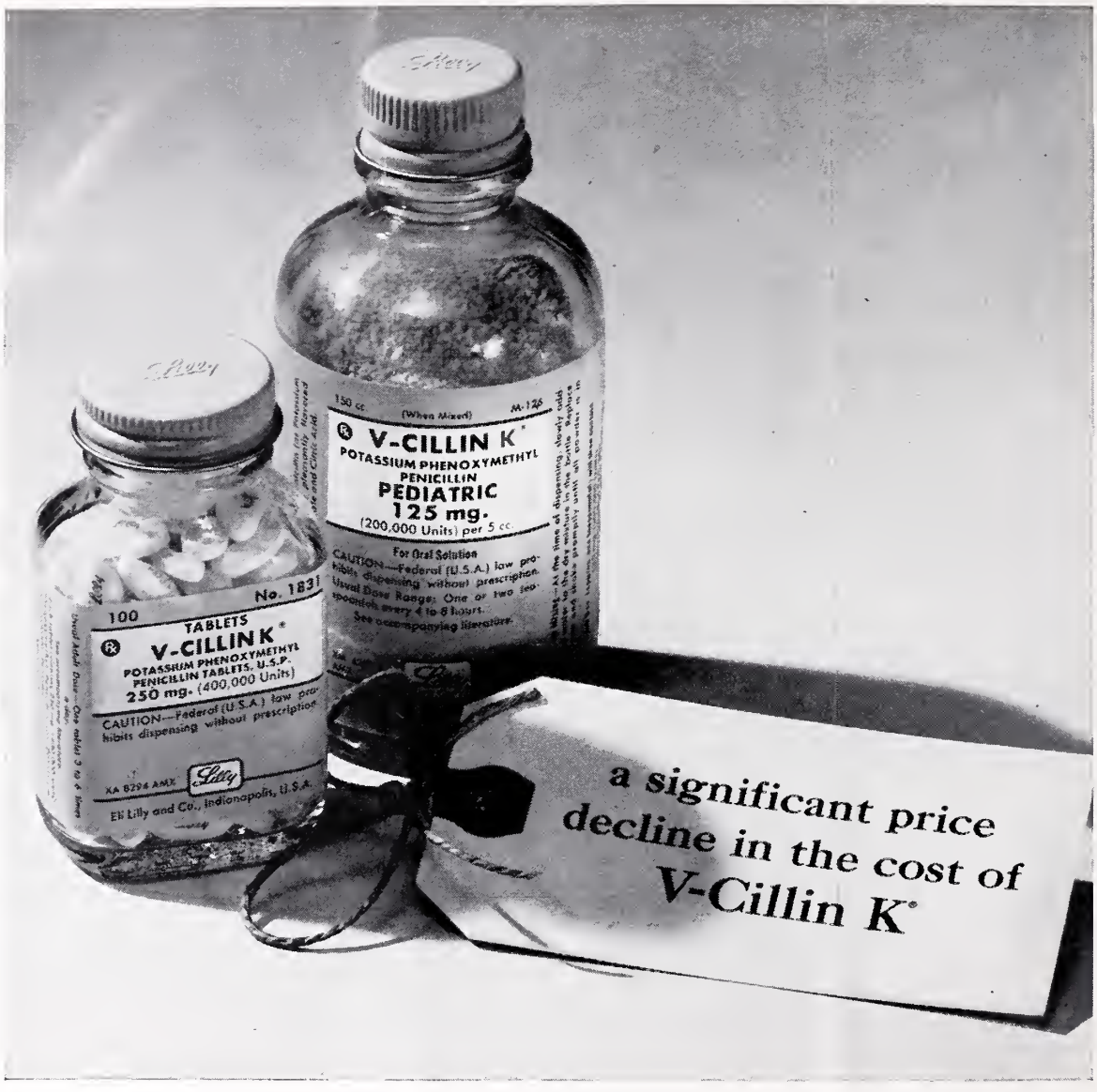
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October, 1965

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Precautions: Lomotil is an exempt narcotic preparation of very low addictive potential: more than three million prescriptions have now been written for Lomotil. Recommended dosages should not be exceeded. Lomotil should be used with caution in patients with impaired liver function and in patients taking addicting drugs or barbiturates.

Side Effects: Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness and insomnia.

Dosage: For full therapeutic effect – Rx full therapeutic dosage. The recommended initial daily dosages, *given in divided doses,* until diarrhea is controlled, are:

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6 to 12 months–4 mg. (½ tsp. q.i.d.)
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Adults: 20 mg. (2 tsp. 5 times daily or 2 tablets 4 times daily) **Based on 4 cc. per teaspoonful.*

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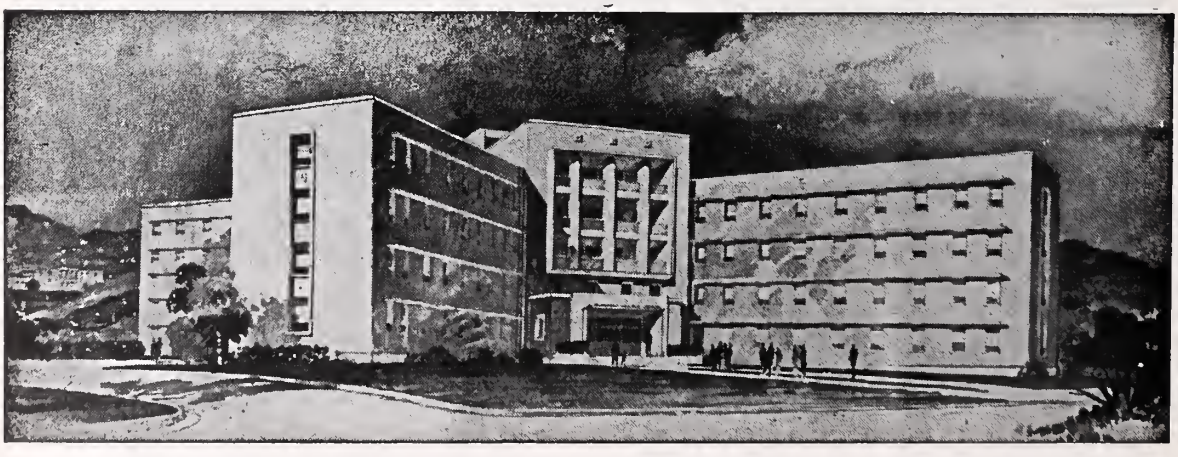
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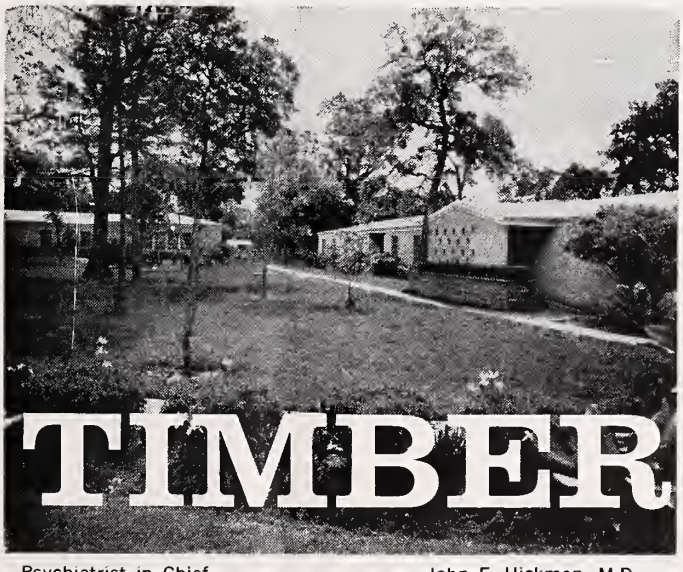
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Lehmann, H. E., Canad. Psychiat. Assn. J. *4(S)*: 1-12, 1959



An antidepressant designed for the clinical realities of office practice

As many physicians have reported, the large majority of neurotic depressed patients suffer from both depression and anxiety. It may be difficult to decide whether these patients are primarily depressed or primarily anxious. And yet drug treatment of only the symptom which seems more prominent may exacerbate the untreated element of the depression complex.

Consequently, it would seem that therapy specifically aimed at both the depression and associated anxiety and tension should increase success in treatment.

This is one of the important reasons why 'Deprol' has proved particularly helpful. For 'Deprol' acts rapidly both to lift the mood and to relieve the associated anxiety, tension and insomnia.

And side effects, at recommended dosage, have been infrequent and generally readily controlled.

Indications: 'Deprol' is useful in the management of depression, both acute (reactive) and chronic. It is particularly useful in the less severe depressions and where the depression is accompanied by anxiety, insomnia, agitation, or rumination. It is also useful for management of depression and associated anxiety accompanying or related to organic illnesses.

Contraindications: Benactyzine hydrochloride is contraindicated in glaucoma. Previous allergic or idiosyncratic reactions to meprobamate contraindicate subsequent use.

Precautions: *Meprobamate*—Careful supervision of dose and amounts prescribed is advised. Consider possibility of dependence, particularly in patients with history of drug or alcohol addiction; withdraw gradually after use for weeks or months at excessive dosage. Abrupt withdrawal may precipitate recurrence of pre-existing symptoms, or withdrawal reactions including, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, the dose should be reduced and operation of motor vehicles or machinery or other activity requiring alertness should be avoided if these symptoms are present. Effects of excessive alcohol may possibly be increased by meprobamate. Grand mal seizures may be precipitated in persons suffering from both grand and petit mal. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side effects: Side effects associated with recommended doses of 'Deprol' have been infrequent and usually easily controlled. These have included drowsiness and occasional dizziness, headache, infrequent skin rash, dryness of mouth, gastrointestinal symptoms, paresthesias, rare instances of syncope, and one case each of severe nervousness, loss of power of concentration, and withdrawal reaction (status epilepticus) after sudden discontinuation of excessive dosage.

Benactyzine hydrochloride—Benactyzine hydrochloride, particularly in high dosage, may produce dizziness, thought-blocking, a sense of depersonalization, aggravation of anxiety or disturbance of sleep patterns, and a subjective feeling of muscle relaxation, as well as anticholinergic effects such as blurred vision, dryness of mouth, or failure of visual accommodation. Other reported side effects have included gastric distress, allergic response, ataxia, and euphoria.

Meprobamate—Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

Dosage: Usual starting dose, one tablet three or four times daily. May be increased gradually to six tablets daily and gradually reduced to maintenance levels upon establishment of relief. Doses above six tablets daily are not recommended even though higher doses have been used by some clinicians to control depression and in chronic psychotic patients.

Supplied: Light-pink, scored tablets, each containing meprobamate 400 mg. and benactyzine hydrochloride 1 mg.

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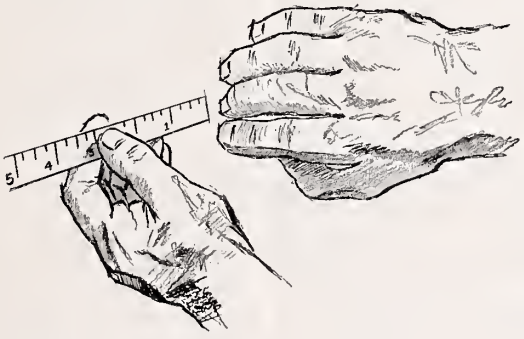
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Dr. Blasingame to Speak on Medicare and Medicine at Southwestern Medical Ass'n Meet

Dr. F. J. L. Blasingame, Executive Vice-President of the American Medical Association, will speak on "Medicare — Its Effect on Your Practice" at the 47th Annual Meeting of the Southwestern Medical Association in El Paso, November 4-6, 1965.

Dr. Blasingame will be the luncheon speaker at a special event on the opening day of the three-day meeting. He will talk at 12:20 p.m. at a luncheon to be held in the Desert Hills Motor Hotel. Wives are invited.

The Southwestern meeting will have the Sheraton-El Paso Motor Inn as its headquarters for scientific sessions and exhibits but the Dr. Blasingame talk has been moved to the nearby Desert Hills Motor Hotel for what is expected to be a large audience.

The Faculty for the scientific sessions will come from the University of California at Los Angeles School of Medicine, and the program will be under the direction of Dr. Charles R. Smart, Assistant Professor of Surgery in Residence and Assistant Director of Postgraduate Medical Education at the School of Medicine.

General subject for the sessions will be Chest Diseases. The program has been approved by the Texas Academy of General Practice. The meeting is open to all physicians. Registration fee will be $15 and the registration table will open at 8 a.m. daily at the headquarters in the Sheraton-El Paso Motor Inn. Scientific sessions will be held in mornings only.

Speakers will be:

Arthur A. Clinco, M.D., Associate Clinical Professor of Psychiatry.

Donald G. Mulder, M.D., Associate Professor of Surgery.

John F. Murray, M.D., Associate Professor of Medicine and Physiology.

Justin J. Stein, M.D., Professor of Radiology and Chief, Radiotherapy Division.

An elaborate program of entertainment for physicians and their wives has been planned. On Thursday morning there will be a brunch and style show at Coronado Country Club. In the afternoon physicians and wives will have a choice of a hospital or sightseeing tour. El Paso doctors' wives will drive guests on the tours. A visit to the Turn of The Century for an oldtime melodrama is on the schedule that night.

Slated for Friday morning is a shopping tour of the Bassett Shopping Center. In the afternoon El Paso doctors' wives will drive guests to the new Camino Real Hotel in Juarez for luncheon. A visit to the Museum of the Arts and Crafts center in the new area of the Border Improvement Program in Juarez will follow. Physicians and wives will then be guests of the Waterfill Distillery in Juarez for a cocktail party and next the Juarez Greyhound Race Track. A tour of the El Paso Museum of Art is scheduled for Saturday morning, with a coffee at the El Paso County Medical Society's Turner Home. Golf in the afternoon or horse-racing at Sunland Park will complete the meeting's entertainment calendar.

Officers of the Association are Dr. Clement C. Boehler, El Paso, President; Dr. Frank A. Rowe, Albuquerque, President-Elect; Dr. Zigmund W. Kosicki, Santa Fe, Vice-President; and Dr. Sol Heinemann, El Paso, Secretary-Treasurer.

Dr. Gordon Black of El Paso is general campaign chairman. Members of his committee are Dr. Heinemann, finance; Dr. A. Robert Nering, program; Dr. Laurance N. Nickey, publicity; Dr. Louis W. Breck, properties; Dr. J. Philip Richardson, exhibits; Dr. Richard J. Harris, hotel; Dr. Billy L. Farmer, golf; Dr. R. A. D. Morton, Jr., transportation; Dr. George W. Iwen, social; Dr. and Mrs. Robert F. Boverie, president's party; Dr. Humberto Quirarte, Juarez entertainment; and Mrs. Louis W. Breck, women's committee chairman.

Exhibitors will be Bentex Pharmaceutical Co., Deseret Pharmaceutical Co., Geigy Pharmaceuticals, Eli Lilly and Co., Mission Pharmacal Co., Norton Bros., Parke-Davis, Sandoz Pharmaceuticals, Smith, Miller and Patch, Inc., Southwest Air Rangers of El Paso, Inc., Southwestern Surgical Supply Co., E. R. Squibb and Sons, U. S. Vitamin and Pharmaceutical Co., West Texas Medical Specialties, Westwood Pharmaceuticals, and Wyeth Laboratories.

Grand Rounds At William Beaumont General Hospital

Gout and Hyperuricemia

Moderator: Major Frank Chamberlin,
Department of Medicine, Lt. Col. Robert H. Moser, *Chief*

I. Clinical Syndrome—Lt. Col. Robert H. Moser, MC
II. Uric Acid Metabolism—Major Jerry M. Earll, MC
III. Genetics and Secondary Gout—Captain Jeremiah J. Twomey, MC
IV. Gouty Nephropathy—Major William P. Nelson, MC
V. Laboratory Tests—Major Robert C. Zurek, MC
VI. Therapy of Gout—Major Robert C. Zurek, MC

I. Clinical Syndrome

(Lt. Col. Robert H. Moser)

A. General

Gout is a venerable medical disease. It was described in animals about 500 years B.C.; Hippocrates appended the term "podagra" or "seizure of the foot" to the acute arthropathy. Perhaps no popular disease has had its clinical manifestations documented so thoroughly, had its pathophysiologic mechanisms so well explored and yet remain so enigmatic and apparently paradoxical. Perhaps no disease of such uncomplicated clinical configuration is so frequently missed in diagnosis or misunderstood in natural history. It is upon this latter area that this portion of the presentation will dwell.

Gout or symptomatic hyperuricemia represents a broad spectrum of clinical manifestations. At the near end of the curve we have less than five per cent of patients who become chronic arthritic invalids with disabling joint deformities. At the far end we have those who may survive eight decades of gout without serious arthropathy, whose metabolic affliction has contributed no greater injury to the cartilage of the joints than to the pinna of the ear. Between these extremes we find the vast majority of patients.

An additional clinical fact, perhaps insufficently appreciated, is the renal component of gout. Doctor Nelson will discuss this in some detail later, but it must be added at this juncture that although we have come to recognize gout through its arthritic manifestations, it is the renal component which is the most frequent cause of death.

B. Incidence

1. Sex—predominately male (95 per cent).
2. Age—onset usually in middle age but may occur at any age (10-80.)
3. Race—widely distributed; less common in Negroes; more common in Filipinos.
4. Social factors—gout may occur at all echelons of society; diet is unimportant except as a contributory factor in disease already present.
5. Heredity—striking familial incidence.

C. Precipitating Factors

1. High purine diet (or any major dietary indiscretion).
2. Tight clothing (gloves, shoes).
3. Surgical procedures (major, minor).
4. Emotional disturbances.
5. Infections (major, minor).
6. Drugs (alkylating agents, actinomycin D, testosterone, thiazides, pyrazinamide, pempidine, mecamylamine (Inversine).
7. Direct trauma.
8. Inclement weather.
9. Other diseases.

D. Classification

1. Primary—familial inborn error of metabolism of purines.

a. Hyperuricemia—present in about 25-75 per cent of blood relatives; percentage who will eventually become symptomatic is unknown.

b. Acute gout—manifested by hyperuricemia and recurrent acute arthritis. Exact cause unknown; precipitating factor—stress.

c. Internal gout—? prophylaxis for rest of life. Colchicine before and after surgery to prevent exacerbations.

d. Chronic gouty arthritis—manifested by:

(1) Incomplete resolution of arthropathy.

(2) Radiograpric changes—osseous tophi and destructive changes.

(3) Tophi—eventually develop in 50 per cent of cases. Result from positive uric acid balance with precipitation of sodium urate in tissues. Tissues must be predisposed in some unknown manner.

2. Secondary—may be associated with:

Polycythemia, both primary or secondary—

leukemias and lymphomas (especially following nitrogen mustard, triethylene melamine, and x-ray therapy of these diseases); pernicious anemia, multiple myeloma, lead poisoning, etc. Hyperuricemia of chronic renal disease almost never gives rise to acute gouty arthritis.

E. Early State: (acute)

1. Sudden onset of severe pain at any time of day or night.
2. Cardinal signs of inflamation, i.e., heat, redness, swelling and pain of metatarso-phalangeal joint of great toe in 60-70 per cent. (podagra)
3. Constitutional effects include headaches, tachycardia, malaise, chills, anorexia, pyrexia (103-104°).
4. Laboratory studies show an increase in sedimentation rate, leukocytosis (10,000-20,000 mm^3).
5. Complete recovery of function after acute attack.
6. Desquamation sometimes is extensive.

F. Pathogenesis of Acute Gouty Arthritis

The pathogenesis of the acute attack of gouty arthritis has been the subject of investigation for decades. The most reasonable hypothesis seems to be one proposed by McCarty. Several phenomenon have been observed which seem to suggest a probably pathogenetic mechanism: (1) intra-articular injection of urate in non-gouty humans and dogs reproduces symptoms of acute arthritis; (2) injection of urate is associated with effusions characterized by intracellular urate crystals and small clumps of intracellular material which surrounded the crystals; (3) this is associated with increased intra-articular pressure and synovial vascularity; and finally (4) there is a decreasing pH of the effusion.

McCarty suggested that hyperuricemic body fluids (miscible pool) caused precipitation of sodium urate crystals in joint spaces. This is associated with inflammation and a marked polymorphonuclear response, which in turn is followed by phagocytosis of the crystals with increasing lactate production and decreasing pH of the effusion. The declining pH precipitates further sodium urate crystals which in turn provokes more inflammation. Thereby, the cycle is perpetuated.

Additional evidence comes from the fact that normal white cells contain peroxidase (two per cent of dry weight). This enzyme is essential in converting uric acid to allantoin. A deficiency of this enzyme may be related to the difficulty in keeping urate in solution.

One further observation affords a possible explanation for the almost magical effect of colchicine in arresting the acute inflammatory arthritis. This may be related to colchicine induced inhibition of polymorphonuclear leukocytes; colchicine impairs normal phagocytosis, which in turn decreases the production of lactic acid by neutrophils, which will preclude lowering of pH in the synovial effusion. Urate is much less soluble at acid pH and may precipitate to perpetuate the cycle.

Phenylbutazone is an effective anti-inflammatory agent; it may act through antagonism to bradykinin (a toxic product released when neutrophils degenerate).

These are pertinent observations and may help resolve some of the pathogenic mystery of the gouty arthritis episode. However, it will not fully answer several questions. Why do only 25 per cent of hyperuricemic subjects develop episodes of acute gouty arthritis? What is the mechanism whereby hyperuricemia may persist for years before an episode of acute gouty arthritis occurs? How can so many diverse and unrelated events precipitate acute attacks?

G. Differential Diagnosis

1. Acute cellulitis
2. Acute rheumatic fever (larger joints)
3. Rheumatoid arthritis
4. Infectious arthritis
5. Osteoarthritis
6. Traumatic arthritis
7. "Collagen diseases"
8. Fibrous cystic dysplasia
9. Endochondromata
10. Thrombophlebitis
11. Sarcoidosis
12. Mycoses
13. Bone atrophy
14. Renal disease from other causes
15. Bone tumors
16. Neurofibromatosis

II. Uric Acid Metabolism

(Major Jerry M. Earll)

Gout has been considered a disease of purine metabolism. Recent work has focused attention on alternative pathways competing with de novo purine biosynthesis for utilization of glutamine. Evidence exists that these patients suffer a deficiency in renal formation of ammonia from glutamine.

The chemical history of gout began about the time of the Revolutionary War when uric acid was demonstrated as a constituent of a kidney stone. Soon thereafter, urates were demonstrated in tophi. Garrod demonstrated increased uric acid in the blood just before the Civil War; Emil Fischer identified uric acid as a purine compound. It is interesting to note that although uric acid appears to be a useless metabolic end-product of purine metabolism in man, it is zealously conserved by the kidney. The metabolically important purines, adenine and guanine, serve as building blocks for nucleic acids and as components of cofactors for many biochemical reactions. In most animals, uric acid is catabolized almost entirely by means of uricase to allantoin, and in many species to urea. This does not occur to any great extent in man.

Uric acid in man is derived from three sources: A. It is the chief end-product of catabolism of endogenous purines; B. It may be produced by conversion in ingested purines; C. It may be synthesized directly from glycine and other single carbon precursors without incorporation into tissue purines. As knowledge of the biochemistry of uric acid has increased it has been possible to study the extent to which lack of these factors contribute to the hyperuricemia of gout. Nucleoproteins are contained in the nuclei and cytoplasm of all living tissues. These can be separated into a protein moiety and a large complex polymer, nucleic acid. The key unit of nucleic acid is the nucleotide, a complex of one molecule of a base (either as purine or pyrimidine, see figure 1.), a pentose (ribose or desoxyribose),

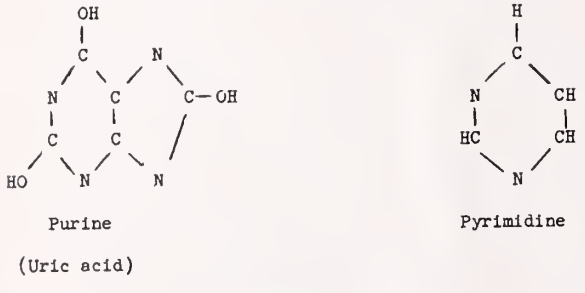


Figure 1.

and a molecule of phosphoric acid. The substitution of uracil for thymine differentiates RNA from DNA. As near as can be determined, pyrimidines are not involved in uric acid synthesis. Purine nucleotides, particularly adenylic and guanylic acid are broken down to uric acid.

The origin of the atoms of a purine ring can be traced to glycine, formate, CO_2, and the common metabolic pool of amino groups. Adenosine triphosphate (ATP) is required to make guanine nucleotides, and guanosine triphosphate (GTP) is required to make adenine nucleotides. This may result in a self-regulating mechanism to maintain the proper ratio of these compounds. Ingested purines are not used as such but are broken down and recycled. Ingested purines are to a large extent converted into uric acid. Both normal and gouty persons on high purine diets increase their excretion of uric acid and transiently increase the serum uric acid. Even with high purine diets, urate genesis from this source does not equal the production from endogenous catabolism. Any factor tending to increase the amount of available amino acids and nitrogen tends to increase production of uric acid. The direct biosynthesis of uric acid from simple precursors probably plays little role in normal persons, although it may be important in the gouty subject.

Intravenous injection of isotopically labeled uric acid results in the estimate that the average male has just a little over one gram of uric acid in his miscible pool. Gouty subjects without tophi have two to four times this amount; the patient with tophaceous involvement may have 15 to 30 times the normal amount. Evaluation of uric acid synthesis has many pitfalls. Increased intestinal uricolysis or deposition in tophi can mask detection of increased production of uric acid by urinary excretion tests. The daily urate turnover is determined by injection of isotopically labeled urates. Normal adult values range between 500-1200 mg uric acid per day. In the gouty subject, there are problems with this test. Tophaceous deposits can produce error because they contribute to the pool being measured. This source of error does not play a role in the patient with the normal values; as many as one third of patients may appear to synthesize normal amounts by this test. Extra-renal excretion pathways are of considerable importance in this group.

A different approach to the problem has been the introduction of isotope labeled precursors to evaluate urate biosynthesis. Maximum excretion of uric acid occurs more rapidly in the gouty subject than in controls and gouty patients with normal parameters of uric acid production. Hyperuricemic individuals are also hyperaminoacidemic and defective cellular transport of both uric acid and amino acids could account for elevated serum

levels. Renal retention of amino acid does not explain the hyperamino-aciduria.

Studies with isotopically labeled compounds support the concept that there is no single metabolic defect in purine metabolism to account for hyperuricemia. Gutman proposes an abnormality in glutamine metabolism as a possible etiology of primary gout. A defect in utilization of glutamine for renal production of ammonia (? glutaminase deficiency) might enhance uric acid production by recycling extra glutamine and possibly influencing a rate determining reaction of purine biosynthesis. It is apparent that we are not yet able to identify the etiology of primary hyperuricemia with assurance. The renal handling of urate may be abnormal and abnormalities of the enzyme amidotransferase and substrate phosphoribosylpyrophosphate have been identified.

III. Genetics

(Capt. Jeremiah J. Twomey)

The gouty diathesis follows an inherited pattern, a precedent established by other metabolic abnormalities (i.e. diabetes mellitus and hemachromatosis). Between 6 per cent and 18 per cent of the relatives of gouty patients develop gout; 25 per cent become hyperuricemic. These figures are influenced by two factors: (1) The low incidence of primary gout among females (one study indicated a four-fold male preponderance; the ratio is more exaggerated in clinical gout), (2) Overall incidence will be less impressive if prepubertal relatives are included in family studies. The emergence of the metabolic abnormality in the post-pubertal years resembles adult diabetes and acute intermittent porphyria. The gouty diathesis is inherited as an autosomal defect since males can transmit it to their male progeny; it is apparently a dominant trait. This leaves the male predominance difficult to explain. The situation is analogous to glucose-6-phosphate dehydrogenase deficiency. Perhaps the defective gene has only partial penetrance and is influenced by a regulator gene located on the X chromosone.

A rare variant of the hyperuricemic syndrome involves children. Hyperuricuria comprises renal function at an early age. Mental and central nervous system dysfunction complete the syndrome. These changes do not appear to result from a direct toxic effect of uric acid on nervous tissue before maturation of an effective blood-brain barrier; there have been isolated reports of primary gout among patients of similar age without neurological complications.

Secondary Gout

(Capt. Twomey)

Gout results from the product of absolute excess of uric acid and length of exposure of relevant tissues. Conditions other than the inherited diathesis for hyperuricemia are potential causes of secondary gout, However, it is unwise to rigidly segregate primary from secondary gout because an underlying gouty diathesis increased the susceptibility for the secondary form.

The secondary variety differs somewhat from the inherited form. (1) Onset is later in life. (2) The incidence is higher in females than in primary gout. Female patients demand a diligent search for an underlying cause. (3) Podagra occurs less frequently than involvement of other joints. (4) Serum uric acid levels usually exceed those of primary gout since this a function of quantity rather than duration of disease. This exposes the kidneys to greater risk.

Hematological abnormalities are the commonest cause of secondary hyperuricemic problems. Diseases in this category include the leukemias, polycythemia vera, myleofibrosis, ineffective erythropoiesis and the lymphomas. In these conditions purine metabolism is greatly accelerated and uric acid production reflects an exhaust pathway. The situation is aggravated by therapy. Six-mercaptopurines impair purine synthesis from xanthines. The accumulation of the latter spills over into uric acid. Alkylating agents accelerate nucleoprotein catabolism, thus increasing uric acid production. Androgen therapy further increases nucleoprotein synthesis in myelofibrosis and increases the probability of secondary gout. The presence of purines in the urine indicates increased nucleoprotein breakdown and has value in distinguishing some varieties of secondary from primary gout.

Secondary hyperuricemia may result from a variety of non-hematological disorders. The hyperuricemia recorded in patients with renal failure is often apparent and may be a reflection of other retained chromogens. Nevertheless gouty arthropathy has occurred in such patients where no evidence of an underlying gouty diathesis or nephropathy existed. The size of the uric acid molecule (mol. wt. 168) makes it difficult to dialyze, and recently gout has seriously complicated some chronic hemodialysis programs. Ketosis is also predisposes to secondary hyperuricemia. This can complicate diabetic acidosis and starvation weight reducing programs. In these conditions

gluconeogenesis and cellular catabolism attempt to compensate. The hydroxybutyric acid formed has been shown to impair uric acid excretion. The same mechanism accounts for the secondary gout that sometimes complicates glycogen storage diseases.

IV. Gouty Nephropathy
(Major William P. Nelson)

It should be remembered that although the arthritis is painful and the tophi disfiguring, it is the nephropathy which is hazardous. Renal failure is the primary cause of death in gouty patients irrespective of age and an important cause of premature death. The renal manifestations in gout are usually so insidious, especially in the early stages, as to be entirely without clinical symptoms and consequently are frequently overlooked. Usually the presence of nephropathy is heralded only by the appearance of albuminuria and a few formed elements in the urinary sediment. It is also well recognized that on occasions nephropathy may precede the first bout of arthritis and the severity of renal damage need not parallel that of articular involvement in any way.

Gouty nephropathy is, in largest part, due to the deposition of uric acid in the kidney—within the distal tubules, collecting ducts, and interstitium. In most cases, the deposits are microscopic, but in an average of 17 per cent of cases uric acid calculi are formed. (Uric acid stones are radiolucent and not visualized unless contrast material is used in the x-ray studies.) The physiologic composition of plasma and most body fluids protects against uric acid precipitation. At normal blood pH (7.4), uric acid is dissociated so that the ratio of salt to acid is 99:1 and total solubility of salt plus acid is approximately 150 mg/100 ml. However the physiologic composition of urine may enhance uric acid precipitation. At a urinary pH of 5 the salt-to-acid ratio is 1:5 with total solubility of only 8 mg/100 ml. The distal nephron is the site of acidification and concentration. It is here in the distal tubule and collecting duct that uric acid precipitation occurs when low pH and reabsorption of water from the fluid exceeds critical levels. (An important therapeutic implication is that administration of alkalinizing salts can increase uric pH to-say-pH 7-and increase urate solubility by a factor of 15 to approximately 120 mg/100 ml.).

Deposition of urates constitutes the *primary* nephropathy of gout (tubular blockage and destruction, interstitial tophus formation, and calculous disease). *Secondary* changes of infection, abcess formation, hydronephrosis, and renal vascular changes contribute. The end result is renal insufficiency and death in uremia in 25 per cent of patients.

A recent series of 28 percutaneous biopsies and 42 autopsies on patients with gout disclosed some previously unappreciated aspects of the renal lesion.

Early changes included a distinct glomerular lesion which the authors considered unique for gout. The black and white photomicrographs were not convincing however. They appeared quite similar to the nonspecific basement membrane lesions of other glomerulopathies (diabetes, lupus, membranus glomerulonephritis). No electron microscopy was performed, unfortunately. Also described were degeneration and pigmentation of the loops of Henle.

Later stages revealed glomerular hyalinization, vascular hypertrophy and tubular atrophy. Interstitial inflammation and fibrosis was considered to be a "nonspecific degenerative phenomenon."

V. Laboratory Tests in Gout
(Maj. Robert C. Zurek)

In the typical case, the diagnosis of acute gouty arthritis can usually be made from characteristic clinical features. Laboratory studies in gout are helpful in supporting diagnosis in the atypical case.

The most familiar and valuable study in evaluating the gouty patient is the serum uric acid level. In most laboratories, a variety of colorimetric methods may be used, all depend on the ability of urates to reduce phosphotungstic acid to a colored compound which is quantitated by the spectrophotometer. Normal values vary with method. For males 5.0-6.5 mgm/100 ml are upper limit values. Normal females will run about one mgm lower. The gouty man will generally have a serum level over 6.5 mgm/100 ml; some 12 per cent of gouty patients will have a serum level below this. Many factors influence serum uric acid; other reducing substances in the blood such as glucose, caffeine, theophillin, etc, may effect the determination; this is generally corrected in the laboratory method. Certain drugs such as low doses of salicylates and thiazide diuretics tend to elevate the serum uric acid. Chronic renal insufficiency, toxemia of pregnancy, and certain blood dyscrasias may also elevate serum uric acid. Uricosuric agents including probenecid (benemid) and phenylbutazone (butazoli-

din), steroids, high dose aspirin (3-6 gm) can lower the serum acid.

The normal 24-hour urinary excretion of uric acid under basal conditions and a low purine diet is generally 400 mgm; 600 mgm/24 hours is evidence of uric acid over-production and may indicate gout.

Presence of sodium urate crystals on microscopic examination of the synovial fluid in the arthritic patient is pathognomonic of gout. Urate crystals may be found only in the leukocytes in early cases, but in chronic cases they are found free in the joint fluid. This observation is not new, but recent reports of the use of the polarized light microscope in detecting urate micro-crystals have increased the yield of this technique. One author states urate crystals can be found in 100 per cent of acute gouty patients.

Analysis of tophaceous deposits for urates is helpful in the diagnosis of the late chronic phase of the disease. They may be present on the helix of the ear, on tendons in joints, and in peri-articular areas or in bones (eye, tongue, buttocks). A sinus may discharge sodium urate intermittently. Any suspected tophus should be examined for presence of urate crystals. The finding of uric acid on analysis of a renal calculus may be the first indication of the gouty state.

X-ray changes of gout are specific and include radiolucent bone lesions in addition to joint and cartilage destruction. These are fortunately late findings and the x-ray is not a useful tool in the diagnosis of early acute gouty arthritis. Soft tissue swelling, with or without evidence of osseous tophi, is usually seen about the metatarso-phalangeal joint of the great toe. Urate tophi usually have no calcium deposited in them, hence, are radiolucent. Radioisotopic measurement of the miscible uric acid pool is presently a research tool. The value for the non-gout patient is 1200 mgm. In gout this is raised many fold.

Renal Complications

Urate gravel or stones are passed by 13 per cent of patients. These are not visible by x-ray. Urate stones do not mean the patient will develop clinical gout; they may appear years before the onset of arthropathy. (Gouty nephropathy has been discussed in a previous section.)

X-ray Evidence

Urate tophi in bones about joints are observed in moderate and advanced cases. They may not be detectible until many years after the first attack.

Laboratory

1. Serum uric acid—usually elevated, especially in chronic stages. Normal level—up to 6 mg/100 cc in males; up to 5.5 mg/100 cc in females. (Confirm normal values in your laboratory.)

2. Urine—excessive urate crystalluria; elevated urinary uric acid excretion. (Normal 400 mg/24 hours, Gout 600 mg/24 hours)

3. Synovial fluid examination—uric acid crystals.

4. Deposits—aspirated tophi; renal calculi (uric acid alone or mixed with other components); miscible pool of uric acid (determined by C_{14} and N_{15} tagged glycine up to 1200 mg in gout.)

VI. Therapy of Gout

(Maj. Zurek)

As for the therapy of gout, this can be divided into three phases:

1. The acute attack.

Colchicine remains the drug of choice. The best result is obtained if treatment is started early. Recommended dose schedules are 0.5-1.0 mgm every 1-2 hours until symptomatic relief is obtained or gastro-intestinal toxicity (nausea, vomiting, or diarrhea) develops. The average dose required is 4-6 mgm. Colchicine may be used intravenously with a dose of 1 mgm and repeated every 1-2 hours with maximum of 4-6 mgm in 24 hours. This route may be useful in the patient with low GI tolerance. Systemic toxicity is rare.

Phenylbutazone (butazolidin) in varying dose schedules (400-800 mgm) in four divided doses for 2-3 days is very effective in acute gout and is preferred by some authorities. Everyone who uses this drug should be aware of its toxicity.

ACTH 60-80 units intramuscularly for 2-3 days or oral steroids are also very effective in aborting the acute attack. Most people reserve these agents for situations where colchicine and butazolidin have failed.

2. The intercritical period.

In the early disease, attacks of acute arthritis are intermittent and resolve without residual. However, 60 per cent experience a second attack within one year and 75 per cent, within two years. As time goes on, attacks become more frequent and protracted. Residual joint disability may ensue. Prophylactic therapy is now recommended by most authorities.

Colchicine daily in low doses (0.5-1.5 mgm) has been reported to be very effective in preventing acute attacks and is rarely associated with toxicity. However, it does not prevent accumu-

lation of uric acid and resulting complications.

The Uricosuric Agents

a. Probenecid (Benemid) in doses varying from 500-3,000 mgm daily in divided doses has been proven to be an excellent prophylactic drug. The goal in this therapy is the maintenance of a normal serum uric acid level. Toxic reactions are rare.

b. Sulfinpyrazone (Anturan) 200-800 mgm in four divided doses daily is a more potent uricosuric agent than probenecid but is more toxic. It may be effective in lowering the serum uric acid when probenecid has failed.

c. Zoxazolamine (Flexin) has been removed from the market because of reports of fatal hepatic necrosis. It was a potent uricosuric drug but had a high incidence of mental depression and GI upset.

d. Phenylbutazone and high doses of aspirin are uricosuric but not recommended for chronic use.

Diet is no longer stressed in the management of the gouty patient. Hemodialysis has been used to lower the serum uric acid in one patient refractory to uricosuric agents. Responsiveness to probenecid returned following four hemodialyses over a 10-day period. A new development in the management of the gout may be in the offing. HPP (4 hydroxyprozolo (3,4d) pyrimid (allopurinol), a xanthine oxidase inhibitor is being investigated. This drug causes a fall in the serum and urine uric acid levels.

It should be pointed out that there is no unanimity of opinion as to the drug or drugs of choice during the intercritical period of gout. Gutman recommends colchicine alone, others suggest uricosuric agents alone. Still others advocate combinations. I personally feel that uricosuric agents are indicated in the presence of elevated serum uric acid.

3. Chronic tophaceous gout

Here all agree on the necessity of life-long uricosuric therapy. This results in mobilization and excretion of urate deposits. Again some include low dose daily colchicine; others reserve it for treatment of the acute attack.

A complete bibliography may be obtained by writing Chief, Department of Medicine, William Beaumont General Hospital, El Paso, Texas 79920.

FROM THE DOCTOR'S LOUNGE . . .

"Nobody Knows The Trouble I See"

Sol Heinemann, M.D., El Paso

Now that the country has gone "all the way with LBJ," American medicine is in for some troubled times. There are those who, like ostriches, have their head in the sand. Many physicians, hospital administrators, and members of the public are afraid to look ahead. Certain things can be foreseen, however, based upon past experience. We in medicine would do well to try to figure out how to live with the monstrosity known as Medicare, as well as determine what we can do to prevent or control the evils that are inherent in the system.

For the next few years the Doctor will be the whipping-boy for all that goes awry in a system of medicine that he did not want. Anything that goes wrong—and there will be many such things—will be placed at his feet. This may include shortage of hospital beds, increased cost of medicine, poor spread of medical care among the population, etc. Within medicine's own ranks one can foresee bitterness. One of the first problems will be the problem of participation. We have already seen this question raised at the 1965 AMA convention. Government medicine, for those over 65, will be the law of the land. While as individuals, doctors can refuse to participate, even to the point of retiring from the profession, small groups of doctors scattered over the country will not have this privilege of breaking the law.

Turn Down Cash Payment?

Either the AMA will lead a nation-wide strike or physicians, *as a group,* will be obligated to participate. Non-participation and civil disobedience will not be countenanced for medicine. If small groups of physicians carry out their threat (that

they will care for the patient and not bill him or sign the forms), one wonders in what hospital they are going to treat their patients. It is a certainty that most hospitals will depend on remuneration from the federal government. No hospital can afford to open its doors to non-participating physicians and thereby become bankrupt. The individual physician could admit one patient under these circumstances; the second admission would be questioned. Another hooker for the physician lies in the fact that many of his patients will be carrying the new government medical insurance to pay the doctor. If the physician should sign these insurance forms he will automatically have signed the necessary forms for the hospital to collect its pay. How many physicians will turn down a cash payment for a principle?

Undoubtedly, with the advent of the bureaucrats, there will be many new rules and regulations. One only has to look at the paperwork, reports and regulations of the Veterans Administration to realize that we are going to see many new rules—in addition to those we have passed to regulate ourselves. It is very doubtful if the AMA will be consulted about these. In addition to the rules that will control the care of patients, drugs used, and reports necessary for billing, we will find that part of the practice of medicine will be under control of Hospital Utilization Committees. Committees such as this have already been formed in many hospitals with a bed shortage. Certainly the only way the government can try to serve all the people wanting hospital beds will be by control of the use of the beds; and here the Utilization Committee will be a necessity. In addition to this there will be local committees to control the building of additional hospitals, etc. These committees will also play a large part in the regimentation of the practice of medicine under bureaucratic control.

In addition to the intramural ill-will that will probably arise with the onset of Medicare, there are other issues that will lead to further division between physicans. This may even lead to their complete defeat through division. When socialized medicine began in England, there was marked difference in the treatment of the specialist and general practitioner. This difference remains. We can look forward to the time when, in addition to a difference in fee schedules, there will be a marked difference in who is allowed to practice in the hospital and who has limited privileges. This probably will contribute still further to who is the Doctor on the inside and who is left out in the cold.

More Regimentation

Last year we saw a report by a presidential committee relating to projected regional complexes for the care and research of stroke, heart disease and cancer. It was recommended in this report that there be about 60 regional research centers and about 360 treatment centers. Perhaps few physicians realize that there are approximately 60 four-year medical schools and 360 Veterans Administration Hospitals in this country. Many of the Veterans Administration Hospitals are connected with medical schools. These V.A. Hospitals are strategically placed and each one could control large areas of practice relating to these diseases. If one visualizes a set-up such as this, one can see further regimentation, with all the hospitals within 100 miles of the treatment center being under the domination of the full-time physicians in the center. This is another aspect of regimentation that may come to pass.

Now that we have looked at some of what physicians may expect in the future, let us look at our hospitals. There is little doubt that we do not begin to have enough hospital beds. In 1959 the Veterans Administration sent a committee to all V.A. Hospitals and made a survey to see if they could be doubled or tripled in patient load just in case the Forand Bill passed. The Forand Bill was the forerunner of our present Medicare.

Our civilian hospitals, which would be faced with overcrowding, can also look forward to bureaucratic control. In addition, they will probably be allowed to dispense only certain lists of drugs such as one sees in the Army and VA Hospitals today. Their overflow will probably be sent to the nearest V.A. Hospital.

Two Classes of Patients

This leaves the hospital administrator faced with running an institution for two classes of patients. One class will be those under Medicare, and the other will be the remainder of the population. Another problem that this administrator will have to face will be the lag in payments resulting in the first six months to a year of Medicare. Very few hospitals today have a financial back-log. Many of them run from month to month and will be hard-pressed because of delayed payments from the Medicare patient. Certainly in the first few months of the system, due

to the bureaucratic handling of funds, the hospital will not be paid immediately. Most hospitals will probably have to borrow money to keep going.

Minimum Wage

One of the greatest dangers that the hospital will face—one that may eventually lead to complete socialization of medicine—will be a minimum wage law. Twice before, Congress has had before it a bill to place hospital employees under the minimum wage of the present $1.25 an hour. Both times this bill has been narrowly defeated. With the advent of the Department of Health, Welfare and Education into the area of hospital care, and with backing by labor unions, it will not be very long before hospitals have to pay the minimum wage and face unionization of their employees. When one realizes that 70 per cent of hospital cost is due to labor, one can see how easily this one fact of minimum wage will lead to the doubling or tripling of hospital cost to the private patient. It should take only a couple of years before the public complains and for the high cost of hospitalization to become a big political issue. One might forecast this campaign issue again for the year 1968, with complete socialization of medicine in the year 1969—three years after Medicare goes into effect! When one realizes that the *estimated* cost of Medicare for 19 million elder citizens is six billion dollars, and that this is with only partial payment of physicians' bills through insurance, one can estimate the cost of socialized medicine at about 60 to 70 billion dollars for hospital care for the entire population. This is bigger than our present defense budget and could lead to nationalization of all industry to pay for it.

In looking into the crystal ball from the standpoint of the public, one must consider how it will respond to the long waiting line to see a physician—the long wait for a hospital bed. Certainly there will be a great deal of competition between people on Medicare and people in the private practice of medicine for available hospital beds. One also wonders what the response of the people will be to the increased taxes that they are going to have to pay to support this system. We have already touched upon the increased hospital cost if the minimum wage law is made applicable, and the public is trained at this point to turn to the government for relief. I am sure that it will do so.

Another problem that will face the medical profession will be the confusion that will exist in the mind of the public as to who their doctor is. Is it the general practitioner on the outside or the specialist in the hospital? Is it the local doctor or the consultant from the medical center who is overseeing all medicine in his area? And what about those taxpayers who live in rural areas where there isn't enough practice to support a doctor? Since they do pay taxes, will they demand that the government send a doctor out to their area? This is a good possibility, since medicine is becoming one of the rights of the citizen according to the socio-political thinking of today.

While the above presentation doesn't cover the entire problem, it gives one food for thought. Should we let the bureaucrats set up this system? Should we demand they use the medical structure that we already have, one which would allow medicine to control and police itself? How can we maintain and promulgate high standards of medical excellence if we don't control this monstrosity? Must we forget excellence in order to produce volume medical care in order to keep the politicians off our back? In any event, the road is going to be long and rough before the practice of medicine is "normalized."

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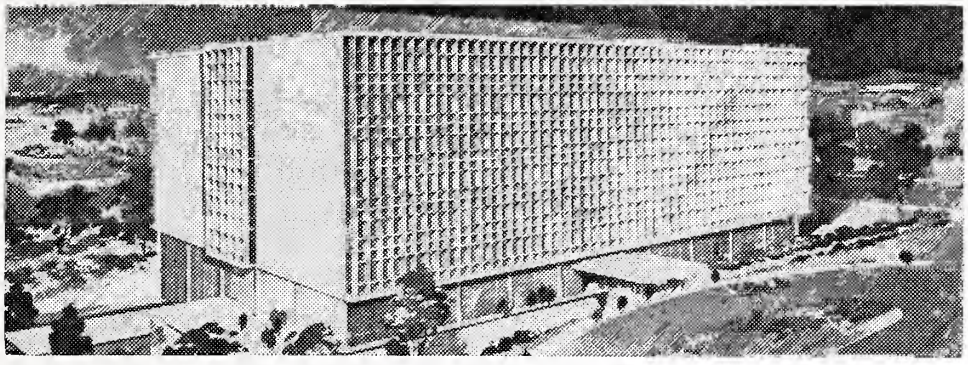
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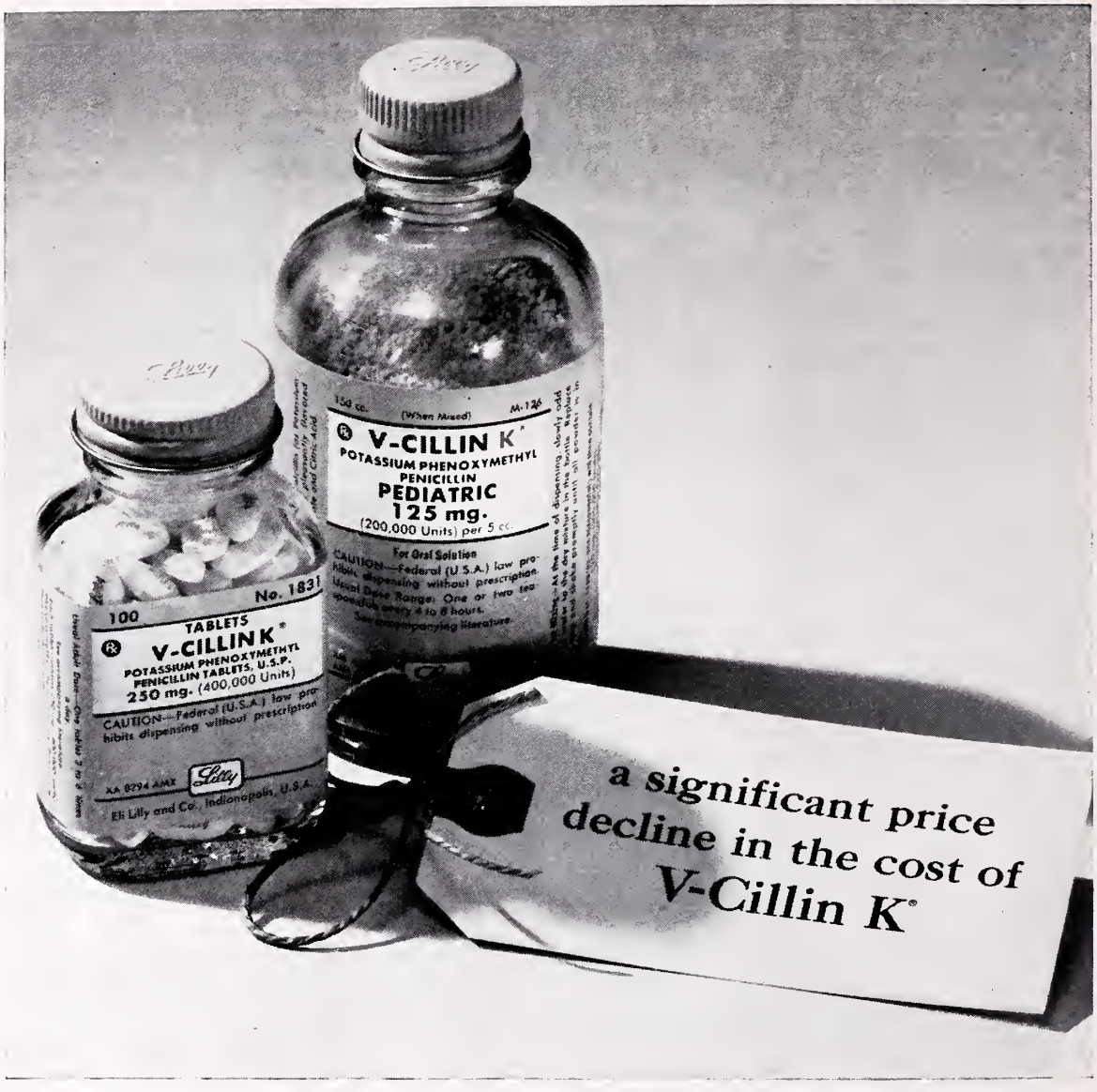
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W. E. Lockhart, M.D., Alpine, Texas

VOL. 46, NO. 11

November, 1965

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Supplied: Tablets V-Cillin K, 125 or 250 mg., and V-Cillin K, Pediatric, 125 mg. per 5-cc. teaspoonful, in 40, 80, and 150-cc.-size packages.

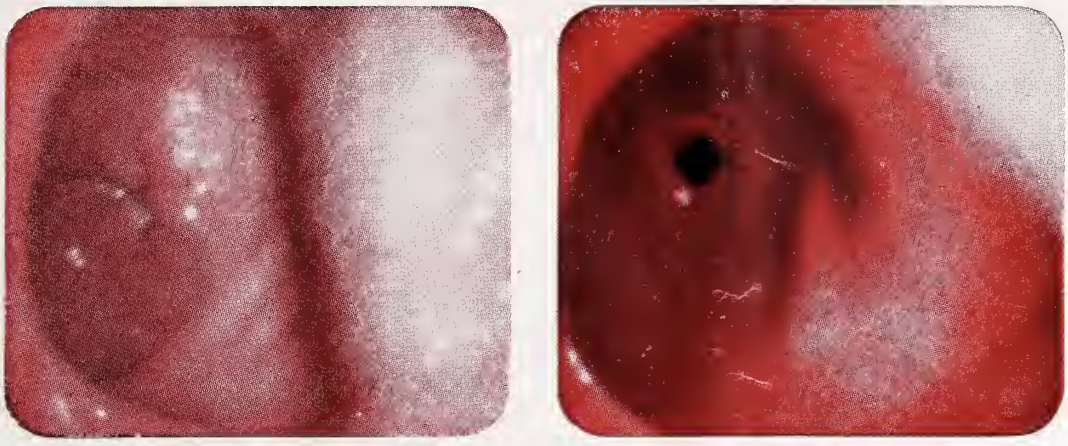
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A/ E. B., male, age 48. Normal antral contraction. Pyloric opening is not seen. It is difficult to differentiate a deep prepyloric contraction from a "pyloric fleurette" or true pylorus.

B/ Same subject after 6 mg. of propantheline bromide intravenously; antral contractions ceased. The pyloric orifice remained open and was easily identified. Better visualization of the antrum was also obtained.

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Pro-Banthine is so effective in anticholinergic action that it may be employed in visualizing the entire pyloric region.

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Pro-Banthine produced complete cessation of gastric, antral and pyloric motor activity with a dose of 6 mg. intravenously. This is approximately one-third the usual oral dose of 15 mg.

Atropine at full normal dosages did not produce such cessation. It required double the usual oral dose of atropine, 0.8 mg. intravenously, to duplicate the aperistaltic action of Pro-Banthine. This dose of atropine produced pronounced discomfort and tachycardia with ventricular rates as high as 150 per minute.

It is this pharmacologic superiority of Pro-Banthine which has made it the most widely prescribed anticholinergic in such conditions as peptic ulcer, functional hypermotility, irritable colon, pylorospasm and biliary dyskinesia.

Dosage—The maximal tolerated dosage is usually the most effective. For most *adult* patients this will be four to six 15 mg. tablets daily in divided doses. In severe conditions as many as two tablets four to six times daily. Pro-Banthine (brand of propantheline bromide) is supplied as tablets of 15 mg., as prolonged-acting tablets of 30 mg. and, for parenteral use, as serum-type ampuls of 30 mg.

Side Effects and Contraindications—Urinary hesitancy, xerostomia, mydriasis and, theoretically, a curare-like action may occur. The drug is contraindicated in patients with glaucoma or severe cardiac disease.

1. Barowsky, H.; Greene, L., and Bennett, R.: Investigators' Clinical Report. Photographs courtesy of Drs. H. Barowsky, L. Greene and R. Bennett.

2. Barowsky, H.; Greene, L., and Paulo, D.: Paper read at Meeting of American Society for Gastrointestinal Endoscopy, Montreal, Canada, May 25-27, 1965.

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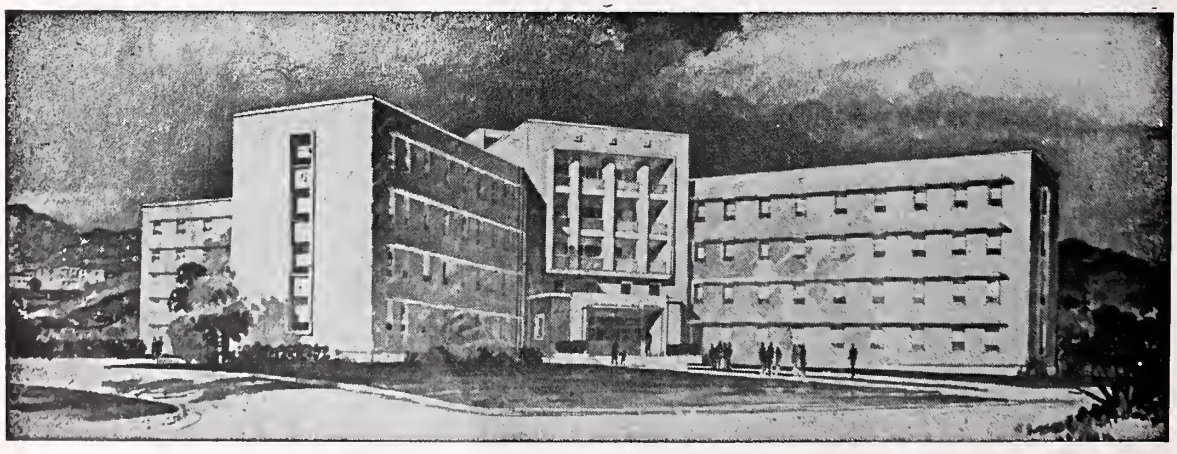
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TEEN-AGE NUTRITION

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More than 40 different chemical elements and compounds have been isolated and identified as nutritional essentials for man. No one food alone can possibly supply all these nutrients in the form and quantity required by the teen-ager. The extremely heavy nutritional commitments of this age group are best fulfilled when the daily diet is selected judiciously from the milk, meat, fruit-vegetable and bread-cereal food groups in the proper balance consistent with individual needs.

Specifically, the milk group serves as a primary source of calcium, phosphorus, riboflavin, high quality protein, and vitamins A and D in the teen-age diet. The group also contributes generous quantities of thiamine, lactose, easily digestible fat, sodium, sulphur, magnesium and trace to moderate amounts of many other nutrients.

This wealth of nutrients makes milk an excellent base on which to build nutritionally complete and appetizing teen-age diets. Available in many different palatable forms, milk and other dairy products provide flexibility in planning the balanced meals needed to satisfy the exacting nutritional requirements of adolescence.

The teen-ager would be hard put indeed to meet the recommended allowances for calcium and to a lesser extent for riboflavin and protein without including at least one quart of milk—or its equivalent in other dairy products—in the well-balanced daily diet.



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"...it is extremely difficult and sometimes impossible to differentiate between 'pure depression' and anxiety and it is questionable whether depression without a certain degree of anxiety really exists."

Lehmann, H. E., Canad. Psychiat. Assn. J. *4(S)*: 1-12, 1959



An antidepressant designed for the clinical realities of office practice

As many physicians have reported, the large majority of neurotic depressed patients suffer from both depression and anxiety. It may be difficult to decide whether these patients are primarily depressed or primarily anxious. And yet drug treatment of only the symptom which seems more prominent may exacerbate the untreated element of the depression complex.

Consequently, it would seem that therapy specifically aimed at both the depression and associated anxiety and tension should increase success in treatment.

This is one of the important reasons why 'Deprol' has proved particularly helpful. For 'Deprol' acts rapidly both to lift the mood and to relieve the associated anxiety, tension and insomnia.

And side effects, at recommended dosage, have been infrequent and generally readily controlled.

Indications: 'Deprol' is useful in the management of depression, both acute (reactive) and chronic. It is particularly useful in the less severe depressions and where the depression is accompanied by anxiety, insomnia, agitation, or rumination. It is also useful for management of depression and associated anxiety accompanying or related to organic illnesses.

Contraindications: Benactyzine hydrochloride is contraindicated in glaucoma. Previous allergic or idiosyncratic reactions to meprobamate contraindicate subsequent use.

Precautions: *Meprobamate*—Careful supervision of dose and amounts prescribed is advised. Consider possibility of dependence, particularly in patients with history of drug or alcohol addiction; withdraw gradually after use for weeks or months at excessive dosage. Abrupt withdrawal may precipitate recurrence of pre-existing symptoms, or withdrawal reactions including, rarely, epileptiform seizures. Should meprobamate cause drowsiness or visual disturbances, the dose should be reduced and operation of motor vehicles or machinery or other activity requiring alertness should be avoided if these symptoms are present. Effects of excessive alcohol may possibly be increased by meprobamate. Grand mal seizures may be precipitated in persons suffering from both grand and petit mal. Prescribe cautiously and in small quantities to patients with suicidal tendencies.

Side effects: Side effects associated with recommended doses of 'Deprol' have been infrequent and usually easily controlled. These have included drowsiness and occasional dizziness, headache, infrequent skin rash, dryness of mouth, gastrointestinal symptoms, paresthesias, rare instances of syncope, and one case each of severe nervousness, loss of power of concentration, and withdrawal reaction (status epilepticus) after sudden discontinuation of excessive dosage.

Benactyzine hydrochloride—Benactyzine hydrochloride, particularly in high dosage, may produce dizziness, thought-blocking, a sense of depersonalization, aggravation of anxiety or disturbance of sleep patterns, and a subjective feeling of muscle relaxation, as well as anticholinergic effects such as blurred vision, dryness of mouth, or failure of visual accommodation. Other reported side effects have included gastric distress, allergic response, ataxia, and euphoria.

Meprobamate—Drowsiness may occur and, rarely, ataxia, usually controlled by decreasing the dose. Allergic or idiosyncratic reactions are rare, generally developing after one to four doses. Mild reactions are characterized by an urticarial or erythematous, maculopapular rash. Acute nonthrombocytopenic purpura with peripheral edema and fever, transient leukopenia, and a single case of fatal bullous dermatitis after administration of meprobamate and prednisolone have been reported. More severe and very rare cases of hypersensitivity may produce fever, chills, fainting spells, angioneurotic edema, bronchial spasms, hypotensive crises (1 fatal case), anuria, anaphylaxis, stomatitis and proctitis. Treatment should be symptomatic in such cases, and the drug should not be reinstituted. Isolated cases of agranulocytosis, thrombocytopenic purpura, and a single fatal instance of aplastic anemia have been reported, but only when other drugs known to elicit these conditions were given concomitantly. Fast EEG activity has been reported, usually after excessive meprobamate dosage. Suicidal attempts may produce lethargy, stupor, ataxia, coma, shock, vasomotor and respiratory collapse.

Dosage: Usual starting dose, one tablet three or four times daily. May be increased gradually to six tablets daily and gradually reduced to maintenance levels upon establishment of relief. Doses above six tablets daily are not recommended even though higher doses have been used by some clinicians to control depression and in chronic psychotic patients.

Supplied: Light-pink, scored tablets, each containing meprobamate 400 mg. and benactyzine hydrochloride 1 mg.

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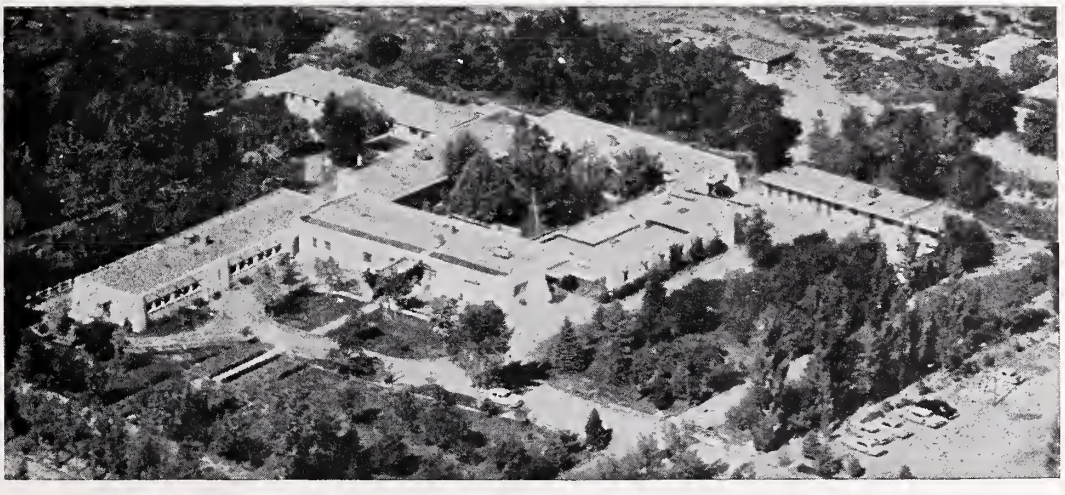
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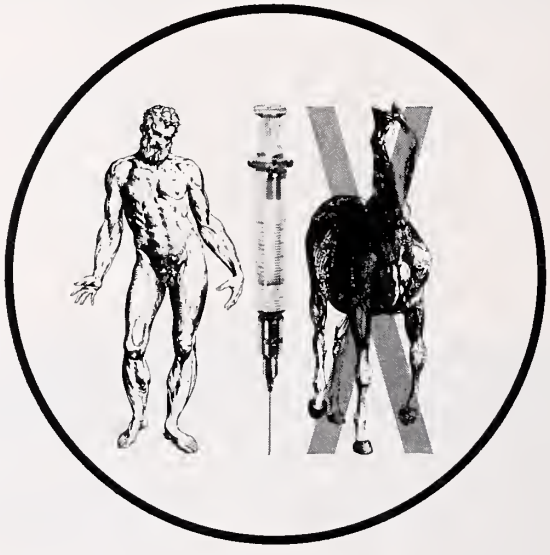
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Side Effects and Precautions: The likelihood of anaphylactoid or serum reactions due to intramuscular injection of gamma globulin is remote. Very rare serious reactions have been reported, however, and their extreme rarity makes it impossible to predict their occurrence. Slight soreness at and over the injection site may be noted. Do not give intravenously. There are no known contraindications.

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References: 1. Rubbo, S. D., and Suri, J. C.: Brit. M.J. *2*:79 (July 14) 1963. 2. Rubinstein, H. M.: Am. J. Hyg. *76*:276, 1962. 3. McComb, J. A.: New England J. Med. *270*:175 (Jan. 23) 1964. 4. Effective tetenus protective level established by Sir Devid Bruce. 6. Editoriel: Tetenus Immunizetion, JAMA *161*:883, 1956.

Clinical Pathological Conference

R. E. Thomason General Hospital, El Paso, Texas

FREDERICK P. BORNSTEIN, M. D., *Editor*

Case #20757, July 30, 1965

Case Presentation and History:
PHILIPP E. BORNSTEIN, *Third Year Student*,
Washington University School of Medicine,
St. Louis, Missouri.*

This 62-year-old Latin-American female (J. U.) was admitted to Thomason General Hospital from Providence Memorial Hospital for the first time on April 30, 1965, with a chief complaint of abdominal distention, jaundice, and mild epigastric pain. The patient died 15 days later.

Six to seven months prior to admission (about September, 1964), the patient first noted epigastric and lower abdominal pain of moderate intensity daily, not related to meals or position. There was some radiation of pain to the back, particularly the right side. This continued for some three months, at which time the patient became aware of the presence of jaundice, generalized pruritus, dark urine, and clay-colored stools. Two months prior to admission, her abdomen became enlarged. There was weight loss of an undetermined amount during this period.

April 24, 1965, she was admitted to Providence Hospital with pain, respiratory difficulty, and inability to move freely because of her distended abdomen. Physical examination at that time revealed a markedly jaundiced, chronically ill Latin-American female in moderate distress with ascites and edema of the lower extremities. Temperature was 98 degrees, pulse 88, respirations 20, and blood pressure 117/62. No evidence of any masses in the abdomen was observed. Heart and lungs were normal.

Laboratory data revealed achlorhydria, bilirubin of 16.5 (8.0 direct; 8.5 indirect), leukocytosis (16,550), alkaline phosphatase 10.7 Bodansky Units; reversed A/G ratio (0.7), with total protein of 7.69 per cent, 4 cephalin flocculation, positive urine bile and stools which were positive for occult blood, but negative for bile.

Dr. William R. Gaddis: Was the test revealing achlorhydria a tube or tubeless type? If it was a tubeless type, it would mean absolute achlorhydria.

(History Continued): EKG on April 25 (done at Providence Hospital) was said to show "myocardial damage including the interventricular septum — probably chronic."

X-ray examination of the gastrointestinal tract revealed a filling defect in the bulb of the duodenum, compatible with duodenal ulcer. There was also diverticulosis of the large intestine. The films were said to show no evidence of malignancy. No cholecystograms or cholangiograms were performed. On two occasions paracentesis was done, and both times cell blocks were negative for tumor cells. The first tap produced 8,300 cc. of clear yellow fluid (Sp. Gr. not done), and a nodular liver was palpable after the paracentesis was completed. On the second tap, 6,600 cc. of icteric amber fluid were withdrawn (Sp. Gr. not done), and on this occasion a large nodular liver was palpated four finger-breadths below the right costal margin, and a "rock-hard mass" was palpated in the liver below the xiphoid. The mass moved with respiration.

In spite of the two paracenteses, the patient's

*Extern in Pathology at Thomason General Hospital.

ascites re-formed, and on April 30, she was transferred to Thomason General Hospital in an essentially unchanged condition.

On admission, physical examination revealed temperature of 97 degrees, pulse 90, respirations 16, and blood pressure of 130/80. This was a well-developed, chronically ill, markedly jaundiced female in mild abdominal distress. The abdomen was markedly distended and ascites was demonstrable, but no masses were palpable. Bowel sounds were increased in intensity. Cardiovascular system was essentially unremarkable; however, one observer reported a few rales at the lung bases and 1 pedal edema. There were decreased breath sounds at the left base. Head, neck, musculoskeletal, genitourinary, and neurologic examinations revealed no significant physical findings.

Past history, family history, and review of systems were non-contributory.

Hospital Course

The first four days after admission were uneventful. She complained only mildly of pain (relieved by Demerol) and she ate poorly. At this time her leukocytosis was still present, prothrombin time was prolonged, and urine was still positive for bile.

On the fifth hospital day, she began complaining of more pain. Intra-venous fluids, antibiotics and steroids were started and a Levine tube was put down. At this time her condition was described as "poor" and from that time on she was semicomatose. On May 6, her blood ammonia was 375 mg. per cent, and her chest films were described as showing a minimal pneumonitis at the left base, possibly secondary to a subdiaphragmatic process. The lungs were otherwise clear and the cardiac silhouette was normal. On May 7, her LAP was 480 units. On the eighth day, her complaints became more pronounced and her condition was termed "critical." On the 12th day her condition was described as poor and her course as "downhill."

On the 15th day she became hypotensive, hypothermic, and she quietly expired on the evening of May 15.

Throughout her hospital course her temperature rose only once above normal, and from the eighth day her temperature fluctuated between 94 and 95 degrees F.

X-Ray Comments

Dr. Vincent M. Ravel: I essentially agree with the x-ray findings as presented; however, I believe that I see in the gallbladder region some opacities which I would interpret as gallstones.

Differential Diagnosis

Philipp E. Bornstein

I considered three main pathologic processes in trying to determine what caused this woman's symptoms. These are toxic, inflammatory, and neoplastic processes.

Inasmuch as there is no history of exposure to hepatotoxins or transfusions, we can exclude hepatotoxicity as a cause of this woman's symptoms — except terminally when she was in liver failure. There is no history of alcoholism or malnutrition, and whatever malnutrition was present in this chronically ill woman was probably secondary to her main disease.

Jaundice occurs as prehepatic, hepatocellular, and obstructive type. Obstructive jaundice can be either intra- or extrahepatic. As there is no evidence of a hemolytic process (she had stable hemoglobin and normal rbc morphology), the patient did not have jaundice of the prehepatic type. The physical symptoms and signs of gradually increasing jaundice with pruritus and the laboratory data of increased bilirubin (direct and indirect) as well as increased alkaline phosphatase (nearly 15 Bodansky units) indicate obstructive jaundice. Intrahepatic cholestasis due to chlorpromazine can be ruled out, as there is no positive history of her having taken this drug. This means the obstruction was probably extrahepatic.

Dr. Dale F. Rector: (Discussing laboratory work) First of all, you see an elevated bilirubin, and you think, is this obstructive jaundice? To me, there are no good laboratory criteria for the type of jaundice, only suggestive data. When the bilirubin gets about 20 mg per cent, one thinks about the obstructive type jaundice. Most of the time, the direct will be higher than the indirect. Usually in a short-duration type of jaundice, the SGOT or the SGPT will all be mildly elevated; whereas in a hepatitis, it will be highly elevated (approximately 1,000).

The alkaline phosphatase becomes elevated when there is irritation of the mucosa of the bile ducts. In long-standing obstructive jaundice there is a tendency for the phosphatase to decrease.

The A-G ratio in this case did not particularly help. It suggests that there is probably some abnormal globulin present. To me, the elevated phosphatase with the bilirubin approching 20, suggests some type of obstructive jaundice in this individual. With the transaminases of 390, 58, one would tend to rule out a hepatitis as a factor.

Philipp Bornstein: The increased levels of the transaminase (SGOT, SGPT), the four plus cephalin flocculation, Thymol turbidity 7.4 units, high blood ammonia, and prolonged prothrombin time on the other hand indicate that there is also a hepatocellular component to this jaundice — due to both cell death and decreased numbers of liver cells. The prolonged prothrombin time is probably an accurate measure of liver dysfunction and not of malabsorption of fat soluble vitamin K, since this patient did not respond to injections of vitamin K.

Ascites occurring in the presence of some sort of liver disease and in the absence of anasarca and a very low albumin implies some kind of impediment of the portal circulation such as thrombosis, invasion by tumor, Budd-Chiari syndrome, cardio-respiratory dysfunction and, most importantly, cirrhosis of the liver. The ascitic fluid in cirrhosis is clear, straw-colored or icteric, has a low protein content and a cell content which is mainly mononuclear.

Inflammatory processes can produce both jaundice and ascites. Inflammatory diseases of the gallbladder can be fairly well ruled out in the absence of colicky pain, chills and fever. Most important, there is no previous history of gallbladder disease. Active hepatitis usually presents different physical and laboratory findings and there is usually much more evidence of liver cell damage. Cirrhosis cannot be excluded in light of the severe recurrent ascites, and the fact that the patient died in hepatic failure.

There are four important entities to be considered in the differential diagnosis of obstructive jaundice. They are chemical cholestasis, sclerosing cholangitis, stone, and neoplasm. The first two have been eliminated.

Cholelithiasis could account for the jaundice and its mode of onset. For example, a stone could be corking the common duct. However, cholelithiasis is more often intermittent in nature, giving rise to distinct variations in the intensity of the jaundice. This was not observed.

I don't think any of the inflammatory processes alone could give rise to all the symptoms of this patient. Cirrhosis, if severe enough, might account for all the symptoms, but this is not likely in view of the palpable masses in the abdomen and the rapid downhill course of the patient.

The disease process most seriously considered by the attending physicians was a neoplasm of some type. This is quite feasible when one considers the mode of progression of her symptoms as well as her chronically ill appearance, weight loss and the fact that she ultimately died.

The sites of primary neoplasms which must be considered are: gallbladder, head of the pancreas, extra-hepatic biliary tree, including the ampulla of Vater, and the liver. I have ruled out benign obstructive lesions mainly on the basis of the enlarged liver and the patient's course.

Dr. George W. Iwen: The important thing to decide is whether or not you are dealing with a benign or malignant lesion. Perhaps there should be some strong indication that the lesion was malignant in view of the ascites. Biopsy of the liver probably should have been strongly entertained at some time. Until you actually prove carcinoma, you are going to be disappointed by assuming such, and discovering at autopsy a benign lesion caused death. There have been striking examples of hepatomas growing up into the right side of the heart and producing ascites on the basis of inferior vena cava obstruction. I think it is a strong possibility in this case. I personally think this is a malignant process, and it is a toss-up in my mind whether it is a head of the pancreas or extension of a carcinoma of the gallbladder. I would have urged somewhat further investigation, perhaps a needle biopsy.

Dr. Dale F. Rector: Speaking about biopsies for this purpose, we had recently two examples of exploratory laparotomy done, and the surgeons felt sure there was an obvious malignancy which we could not confirm microscopically. If one goes to the trouble of opening the abdomen, I think it is well worth another five or 10 minutes needed for the frozen section to be sure the tissue is of diagnostic value.

Philipp Bornstein: Metastases from any other distant site can't be ignored as possibilities. The evidence for another primary is lacking, and a completely silent primary lesion is improbable. Also, direct spread and/or metastases from any of the sites mentioned previously, to any other of these sites is possible and has to be considered.

Carcinoma of the gallbladder is not infrequent — comprising 4 per cent of malignancies. It is more frequent in females than males in a ratio of three or four to one, and occurs mainly during the sixth and seventh decades. There are two main types: papillary and infiltrating — the latter produces a hard mass and comprises about 70 per cent of the tumors. About 90 per cent of these carcinomas are adenocarcinomas. Squamous cell carcinomas are rare. The tumor spreads both by direct local invasion and via lymphatics, and can

produce obstructive jaundice both ways. Survival is six months to one year from onset of symptoms.

All the symptoms and most of the lab data — especially those pointing to the obstructive jaundice — are compatible with such diagnosis. The rock-hard mass palpable below the xiphoid could well be such a tumor, and liver enlargement is compatible with direct or metastatic spread.

Courvoisier's law should be mentioned here, as it is traditional to apply it in such cases. It states that when complete obstruction is due to tumor, the gallbladder is distended and palpable. When it is due to a stone, the gallbladder is not palpable, because gall stones are so often associated with a thick, contracted gallbladder. In view of the palpable mid-epigastric mass which may be the gallbladder, it does seem that this law may hold. Most texts state that this rule is valid in only 50 per cent of cases.

Carcinoma of the gallbladder is associated with stones in 80-95 per cent of cases. We now have x-ray evidence for the stones, although they can certainly be silent. I think that this is important in considering whether the gallbladder is the primary site of the lesion or is only involved in a metastatic process from the other sites mentioned.

Ascites is fairly infrequent with carcinoma of the gallbladder — occurring in only 20 per cent of cases as compared with over 60 per cent in carcinoma of the liver. When ascites does occur, it is often a neoplastic peritonitis. The fluid withdrawn from the patient's abdomen is more suggestive of an ascites due to cirrhosis than due to such a peritonitis. Metastases to lymph nodes producing compression of the portal vein could produce such ascites, as could direct invasion of the portal system by the tumor. In both cases, splenomegaly (whether palpable or not) might be expected. This was not observed in this patient.

Carcinoma of the head of the pancreas is more common in males than in females, the ratio being two or three to one. It occurs most often in the sixth decade. It is associated with obstructive jaundice, and in addition can produce an altered glucose tolerance test or even frank diabetes. Changes in serum lipases, amylase, as well as decreased duodenal secretions are seen. We have no evidence for such changes.

In the absence of fat or undigested protein in the stools, the clay color speaks more for the absence of bile than for a deficiency of pancreatic enzymes.

The increased LAP (leucine-aminopeptidase) is compatible with such a neoplasm, but the values are usually higher (up to 1270 units). The observed value of 480 units in this case is compatible with any neoplasm of this region and is not really specific.*

The x-rays showed a deformity in the duodenal bulb which may have been caused by a tumor. Two recent cases of carcinoma of the head of the pancreas which I studied at this hospital had such a deformity of the duodenum on x-ray. Neither had marked ascites. In any case, the ulcer crater may account for the presence of occult blood in the stool.

Carcinoma of the extrahepatic bile ducts occurs in the age range 60-70, is more frequent in males than females (the ratio is two or three to one), and produces early progressive jaundice. If located primarily around the ampulla of Vater, sloughing and necrosis of the tumor may occur, and this results in an obstructive jaundice which waxes and wanes. The time of survival is about six months from onset of symptoms.

In this case, the obstruction was without remission, and this makes such a diagnosis less likely, although the possibility that there were some unreported episodes of jaundice cannot be ignored. When the patient was admitted, the tumor may have been well implanted and produced the obstruction without any remissions. Ascites does not form a prominent symptom in this neoplasm.

This patient's liver presents a complex of symptoms and lab data which I feel cannot be fully explained on the basis of a neoplasm alone. The nature of the ascites, the reversed Albumin-globulin ratio, and the other lab data compatible with hepatic dysfunction, to me indicate cirrhosis. We should not be put off as easily by her lack of any history of liver disease as we are by her lack of gallbladder disease. She could easily have had a subclinical, asymptomatic case of mild hepatitis in the past which resulted in a cirrhosis. The addition of a neoplasm to such a compromised liver could then produce the present clinical picture.

The commonest malignancies of the liver by far, are metastatic, and metastases from any of the sites mentioned or direct spread could explain the large, nodular liver. Saphir states that metastases to a cirrhotic liver are rare — so, if the masses palpated in this liver are tumor, they probably are either primary carcinoma or result from direct invasion from a nearby primary.

Primary carcinomas of the liver are of two main types: hepatic cell carcinomas, and cholangiocarcinomas. The incidence in greater in

*Reference: Sigma Technical Bulletin #250 (Rev.) 1963.

males than in females; the commonest age being from 40-60. The hepatic cell variety is the commoner of the two, comprising 90 per cent of primary liver carcinomas. Of these, 50 per cent are preceded by some form of cirrhosis. Commonly associated phenomena are: an enlarged, nodular liver, reversed A/G ratio, icterus, abdominal pain, weight loss, and edema of the lower extremities. Cholangiocarcinoma is a rarer tumor somewhat firmer, and about one-third of the cases are associated with portal vein thrombosis which leads to ascites.

If the ascites is due to cirrhosis, then the whole picture is compatible with a hepatoma. The incidence of these tumors is quite low, being only 0.03-0.3 per cent of primary malignancies.

Most of the other lab data is fairly non-specific. The high white count, and remarkably stable hemoglobin have puzzled me. Perhaps the pneumonitis at the lung base found on both x-ray and physical examination could explain the leukocytosis. Pneumonia is fairly common in terminal cancer patients. In spite of the presence of occult blood in the stool, the hemoglobin remained pretty constant — perhaps there was some dehydration which produced a false high result, or perhaps there really was no significant blood loss.

The pain described was not very specific and does not fit any one of the neoplasms better than any other. This may well have been due to stretching of Glisson's capsule over the enlarged liver.

Summary

Even after considering all the possibilities, there are certainly some which I've left out and should have included. The picture isn't particularly clear. This patient probably died of one of four possible malignancies and had an associated cirrhosis of the liver. Statistically, the most likely tumor is carcinoma of the gallbladder with extension to the liver and perhaps into the rest of the extrahepatic biliary tree. While a primary neoplasm of the liver (in association with cirrhosis) presents a neat picture all localized to one organ, it is 10-15 times less common than carcinoma of the gallbladder. Carcinoma of the head of the pancreas seems least likely of the four in the absence of enzymatic changes usually associated with this tumor. Carcinoma of the extrahepatic tree is also a less likely possibility because of the unremitting nature of the jaundice, and because it is a rare tumor.

My final diagnoses are:

Carcinoma of the gallbladder with direct invasion of the liver and metastases to bile ducts.

Cirrhosis of the liver.

Pneumonia.

If I had a second choice, I think it would be a hepatoma.

Dr. William R. Gaddis: Dr. Ravel, in view of the displacement of the organs, could you anticipate metastases perhaps from the stomach?

Dr. Vincent M. Ravel: She certainly could have had.

Dr. William R. Gaddis: Carcinoma of the stomach has been overlooked often by radiologists, particularly if they are in the fundus. They don't always show up.

Pathological Discussion

Frederick P. Bornstein, M. D.

On autopsy I found a rather obese, elderly woman. The body was profoundly jaundiced, and the abdomen markedly distended. Internal examination again revealed marked generalized icterus and marked ascites. 5,000 cc. of ascitic fluid were removed from the peritoneal cavity. The main lesions were found in the liver and biliary system. The liver weighed 1,700 grams (Figure 1). The liver was dark green, granular



Figure 1: Obviously cirrhotic liver with large tumor mass in center.

and showed an obvious cirrhotic pattern on the surface. Within these cirrhotic areas there were a number of tiny, grayish white tumor nodules. In addition, the medial half of the right lobe was taken up by a bright yellow tumor mass which measured about 6 x 6 x 8 cm. and which was firmly attached to the transverse colon. However, there was no lesion in the colon. On sectioning through this tumor, I found several compressed large bile ducts and markedly dilated bile ducts through the tumor free areas. However, a gallbladder could not be demonstrated. Cystic and

hepatic ducts were easily demonstrable and distended. Therefore, the compression of the biliary system was intra- and not extra-hepatic. In addition, there was a moderate degree of gastro-intestinal hemorrhage, probably secondary to the jaundice.

Following the gross examination I made a preliminary diagnosis of cirrhosis of the liver with primary hepatic carcinoma, mainly because these two conditions go together very commonly, and there was no demonstrable gallbladder available for examination.

On Microscopic Examination (Figure 2) I had to change my opinion. The microscopic examination reveals a typical mucinous adenocarcinoma which is obviously of a type which does not arise from liver parenchyma but can arise from the biliary system and probably arose in the gallbladder. By the time the patient died, the gallbladder had been completely destroyed by this carcinoma. Final anatomical diagnoses in this case are therefore:

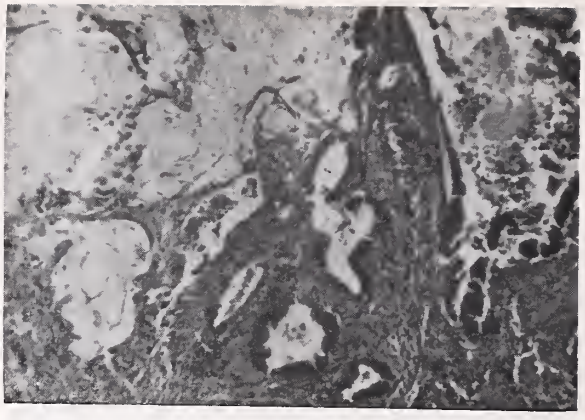


Figure 2: Mucinous adenocarcinoma, low power magnification.

Mucinous adenocarcinoma of gallbladder with invasion of liver.
Compression of biliary system.
Cirrhosis of the liver.
Icterus.
Ascites.

Laboratory Findings

CBC	4-25-65	5-1-65	5-5-65
W.B.C.	16,550	23,000	22,600
Neut.	76	80	80
Bands	4	8	7
Juveniles	—	1	1
Lymphs.	20	10	5
Monos.	—	1	7
Eosins.	—	—	—
Hb.	13.5	13.2	14.1
Hct.	42	44	45

Liver Function Tests

1. **Alkaline Phosphatase**
   4-24-65 10.7 Bodansky Units
   5-7-65 14.5 Bodansky Units
2. **Transaminases**
   4-25-65 SGPT 58 units.
   5-7-65 SGOT 390 units.
   5-7-65 SGPT 80 units.
3. **Prothrombin time**
   4-25-65 16.5″ (13 control)
   5-6-65 19.0″ (13 control)
   5-7-65 18.0″ (13 control)

Three stool specimens were clay colored, positive for occult blood, negative for bile, showed traces of urobilinogen, and no ova or parasites were noted.

Pap Smear: Neg. 4-25-65
Paracentesis: 4-25-65
W.B.C. 920 mm^3
R.B.C. 2,520 mm^3
Differential:
68% Lymphs.
32% Segs.

Pity the Poor Utilization Committee*

The authors of HR 6675, mindful of the tendency of human beings to abuse anything which they consider "free", have cast the physician in the role of a policeman. It provides that medical staffs of hospitals shall develop utilization committees to control overutilization of hospitals and physician services. It even stipulates how such committees shall be established in the event the hospital staffs fail to provide them.

Certainly there will be no rush by hospital staff members to obtain places on these utilization committees. Ignoring for the moment the obvious fact that HR 6675 will give the Federal Government a measure of control over even the most remote community hospital, membership on utilization committees will no doubt prove to be the most onerous of the many tasks that staff members must accept. At first glance assignment to such a committee seems to ask too much of the most public spirited physician. If he carries out his duties conscientiously he could be forever condemned to political oblivion both by his irate medical colleagues and the lecherous relatives of the inevitable minority of senior citizens who will try to impose upon the plan. If he is not conscientious the results could be disastrous.

Who, then, should be appointed to utilization committees? The most cantankerous members of the staff, who, if they ever had any political ambitions abandoned them years ago? Or should the quiet ones who attend staff meetings regularly but make their presence known only by the occupation of chairs, never by offering anything constructive or otherwise? We do not recommend either of these classes. Early in the program, particularly, great care should be exercised in the selection of utilization committees and they should be composed only of outstanding leaders among the profession with well developed senses of responsibility. Our model committee member must also have more than his share of tact both in dealing with his colleagues and with the public. Another desirable quality is a thick hide as he will be exposed to slings and arrows from many quarters.

But our utilization committee member may find some consolation in a recent publication by the Blue Cross of New Jersey. This listed the average length of hospitalization for many surgical procedures. It was designed as a guide to committees which might be established to prevent overutilization of hospital insurance. We were pleasantly surprised at the liberality of this list and gratified to learn that for almost every procedure the average length of hospitalization in our institution was from 30-50 per cent less. While we realize that for many reasons the aged and welfare patients always require more hospitalization than the average, the New Jersey table might be used as a rough guide.

But regardless of their capabilities the members of the utilization committees will encounter many difficulties. To paraphrase A. A. Milne, "A policeman's life is terrible hard." — R. C. D.

*Reprinted from the New Mexico Medical Society's NEWSletter, July 30, 1965.

Management of Sprained Ankle in Athletics

W. E. Lockhart, M.D.,* *Alpine, Texas*

Sprained ankle is the most frequent athletic injury that takes a player out of the game, while internal derangement of the knee is the most frequent permanently disabling injury. Among the causes of sprained ankle are the necessity to abruptly change course with cleats on shoe soles that provide an unnatural traction. Sprained ankle is poorly managed in most instances, and this is very costly to the success of the team not alone from the immediate loss of a valuable performer but because poor management may deprive the athlete of his speed and performance in the weeks or months that follow the injury.

In considering the logistics of an athletic season a wise coach will train his men to make known injuries immediately so that proper care can be given that will return the athlete to the game in top form at the earliest moment. Team mates, some of whom will become coaches, should develop a realistic attitude toward injuries. On present day teams with stress on winning there must be in almost every instance a replacement whose performance could certainly excel that of an injured star, and it is good sportsmanship to give the "sub" a chance. A strong competitor must overcome his aggressive tendencies for the moment in favor of the total performance of the team in the present and subsequent games of the season.

When an athlete knows that he has sprained his ankle, he should get off that ankle immediately and not "try it out" by limping around heroically on the field determined to stay in the game. Team mates should assist him to the sidelines to avoid walking on the injury and to avoid a certain embarrassment in being carried off the field on a litter.

On the sideline at the game the team doctor is called upon to make a decision which is very difficult and which time and procedures (especially X-ray film) are wanting. Is this injury mild enough for the player to return to the game? The injured ankle should (1) be kept off the ground — should not be walked on, (2) it should be elevated above the level of the chest — the player should lie down, (3) the ankle should be firmly wrapped with porous, elastic adhesive bandage and (4) the entire foot and ankle should be packed in an abundance of cracked, fresh-water ice held in place by a sheet of plastic.

If the decision is made — wisely or otherwise — to return the injured player to the game, the ice is removed and strips of NON-elastic adhesive tape are applied on top of the elastic. This should NOT be the common "figure of eight" around the foot and ankle. Without applying detergents and without shaving an "anchor" strip of non-elastic adhesive circles the foreleg about 10 inches above the ground. Several one inch strips of non-elastic adhesive are applied from the "anchor" under the sole of the heel to the "anchor" on the other side of the foreleg. The foot should be held in moderate extension so that the player will be slightly "on his toes," and the

*Team Doctor, Sul Ross State Lobos, Sul Ross State College, Alpine, Texas.

one inch tapes should be a bit more tight on the side of the injury, whether inversion or eversion of the foot. The return to the game should be considered at best a risk and a test, and it may be necessary to reverse the decision.

If the decision is to remove the player from the game, all efforts should be made to expedite the speediest of recoveries. The ice can be removed in 30 minutes or an hour, but the elastic adhesive should remain in place. The injured player should immediately be placed on crutches, and the foot should be kept off the ground, and movement should be avoided. As soon as possible X-ray film should be made. If this indicates fracture of the slightest degree of dislocation, the player should be hospitalized immediately and placed under the care of a doctor with orthopedic capability. Details of orthopedic treatment of ankle injuries are not considered in this paper.

If the X-ray film indicates absence of fracture or dislocation, the player may be treated on crutches. The firm elastic adhesive is left in place with careful observation for possible vascular obstruction from excessive swelling inside the bandage. Codein and aspirin is still the best pain reliever, and the dose should take into account the severity of pain, the size of the man and the fact that athletes in general are very sensitive to pain. As soon as possible he should get to bed with the ankle elevated on a pillow. Next day he should be seen by the doctor, who may prescribe a proteolytic enzyme, which may hasten absorption of blood from the torn tissues. The most important thing is to remain on crutches and avoid movement or weight-bearing for at least three days. Resumption of activity should be on the decision of the team doctor.

Treatments mentioned for purposes of condemnation are: (1) The injection of local anesthetic agents into the site of injury with return of the athlete to the game. This should not be done. (2) The use of ultrasound in the convalescent phase of treatment or otherwise. Ultrasound causes molecular disintegration of tissues. especially bone, and does not hasten the healing process but rather delays it. Ultrasound should not be used in the treatment of a sprained ankle. (3) The use of counter-irritants to the skin do no good, do not hasten healing and may cause blisters and possible infection in the skin with increased disability. (4) Excessive use of infra-red heat lamps should be avoided for the same reasons. After three days active movement in a warm whirlpool bath is beneficial under supervision by the team doctor.

El Pasoan Guides American Fracture Ass'n.

Dr. W. Compere Basom, El Paso, serving this year as President of the American Fracture Association, presided at the annual meeting of the organization October 25-28, 1965, in Las Vegas, Nevada, at the Riviera Hotel.

The Association is composed of 450 physicians from Canada, U. S., Mexico, Central and South America. Dr. Basom's term as President is for three years and he will continue in office until February, 1968. The 1966 meeting will be held in Caracas, Venezuela, November 1-6, and the 1967 meeting in Chicago.

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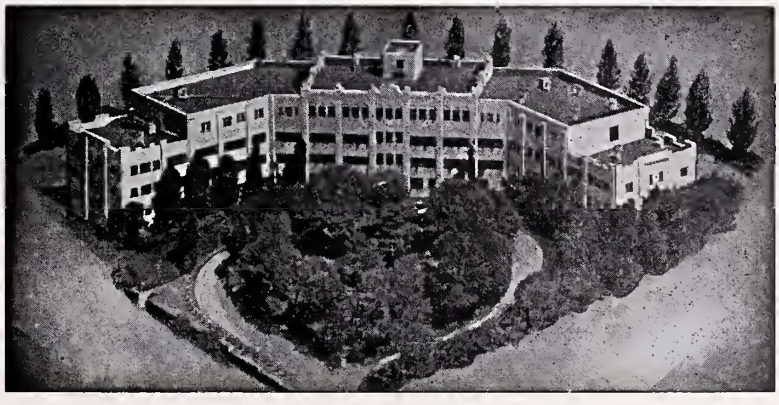
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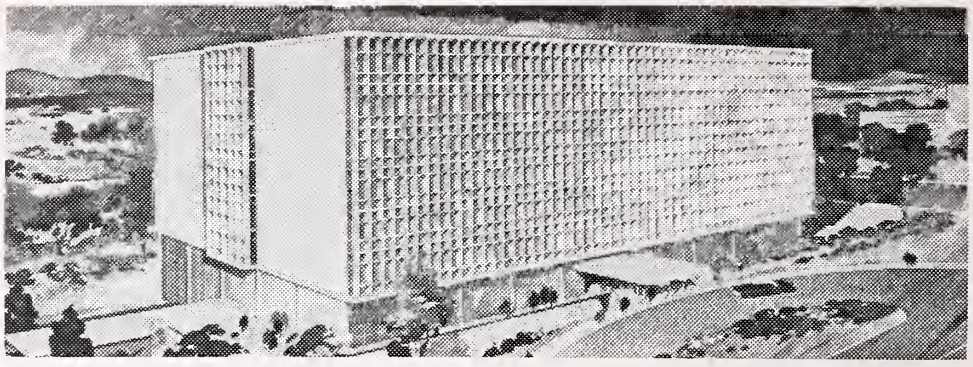
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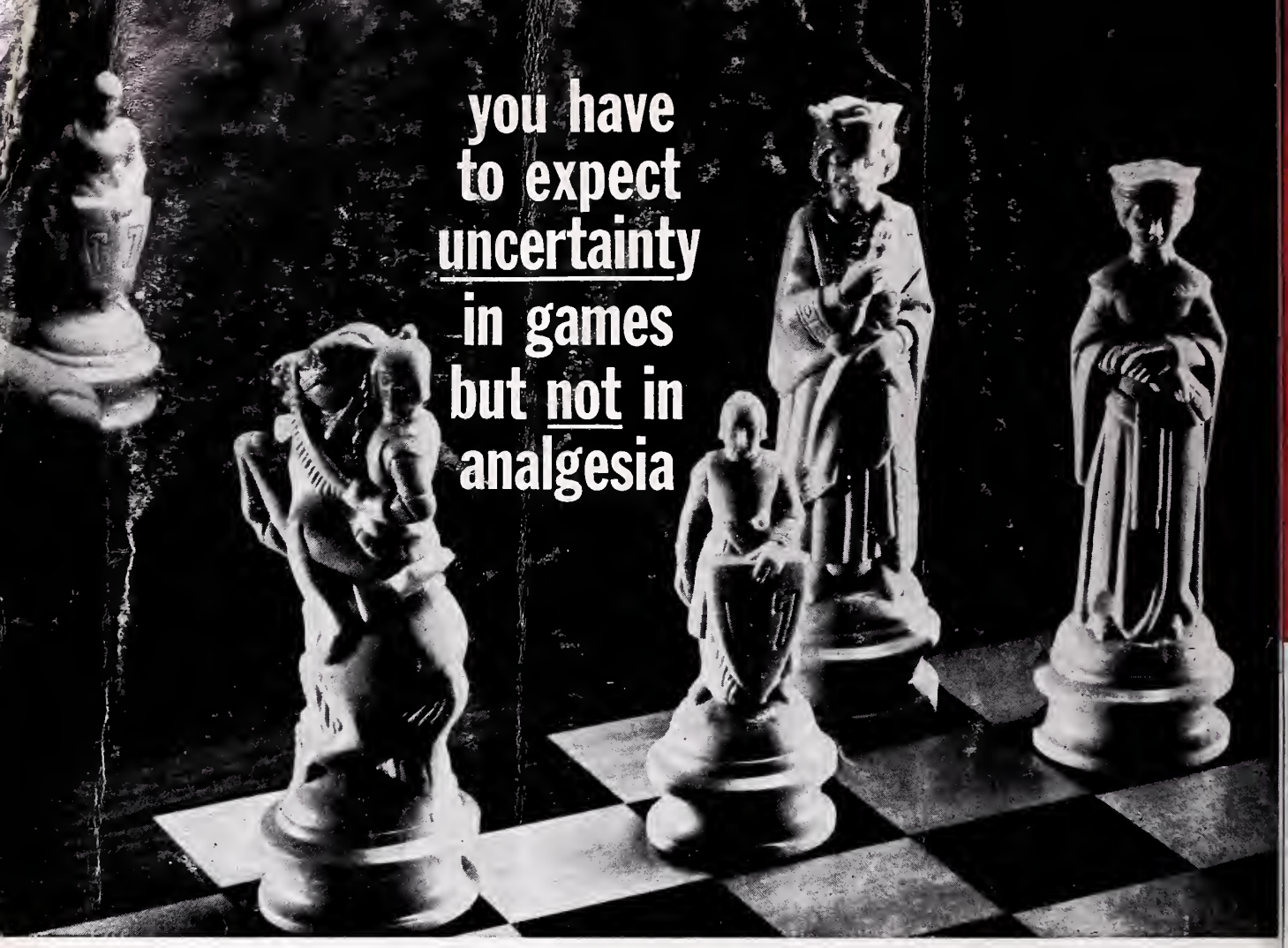
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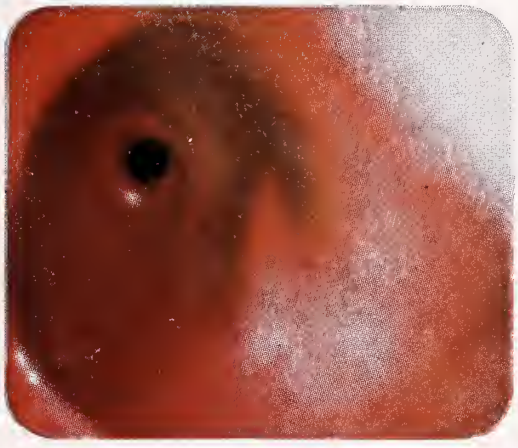
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1. Barowsky, H.; Greene, L., and Bennett, R.: Investigators' Clinical Report. Photographs courtesy of Drs. H. Barowsky, L. Greene and R. Bennett.

2. Barowsky, H.; Greene, L., and Paulo, D.: Paper read at Meeting of American Society for Gastrointestinal Endoscopy, Montreal, Canada, May 25-27, 1965.

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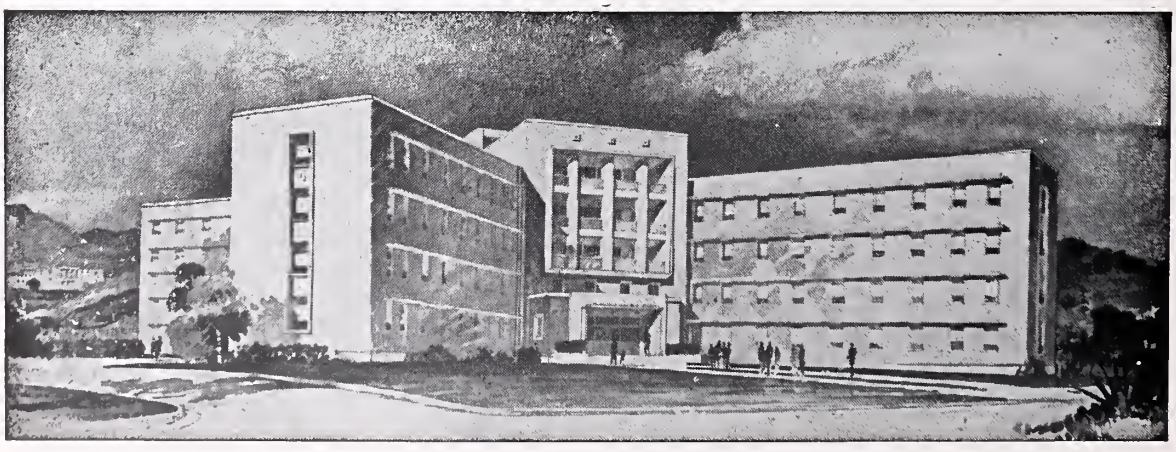
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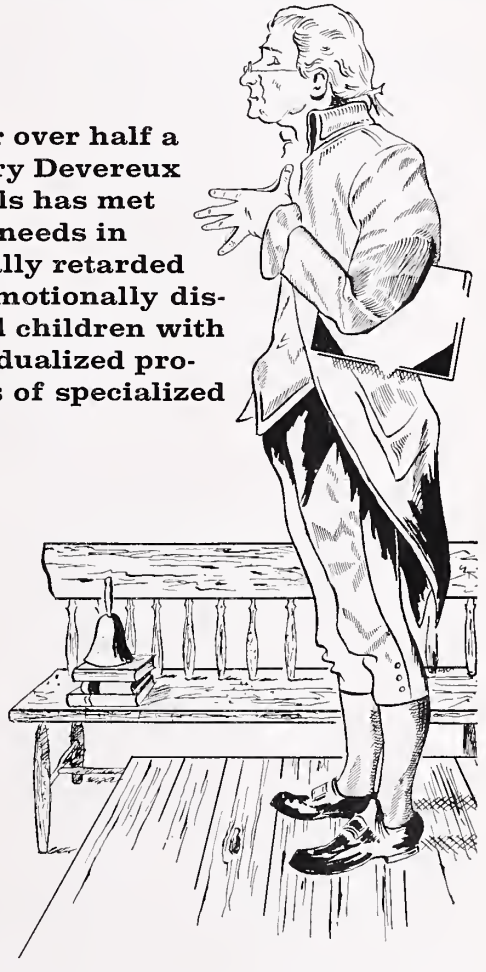
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Coming Meetings

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27th Annual Southwest Allergy Forum, Plaza Motor Hotel, El Paso, Jan. 20-22, 1966.

District I meeting of the TMA, Hilton Inn, El Paso, Feb. 5, 1966.

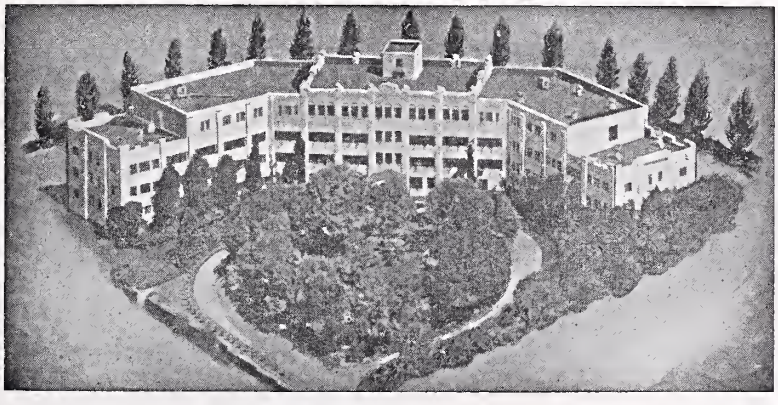
31st Midwinter Clinical Session of the Colorado Medical Society, Brown Palace Hotel, Denver, March 1-3, 1966.

1965-66 Psychiatric Seminar, sponsored by the New Mexico Medical Society, New Mexico Chapter, AAGP, New Mexico State Hospital, University of New Mexico School of Medicine, State Hospital, Las Vegas, New Mexico, March 4-5, 1966.

20th Annual Symposium on Fundamental Cancer Research, University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, March 7-9, 1966.

1966 Arizona Chest Disease Symposium, Ramada Inn, Tucson, March 26-27, 1966.

99th Annual Session of the Texas Medical Association, Austin, April 14-17, 1966.



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Peripheral Nerves of the Forearm and Hand — Their Repair and Regeneration

Morton H. Leonard, M.D., *El Paso*

Anatomy

A knowledge of the distribution of the peripheral nerves of the forearm and hand is essential to make an adequate diagnosis, as well as to find the nerves when they must be repaired. The median nerve enters the forearm between the two heads of the pronator teres and runs sandwiched between the flexor digitorum sublimis and the flexor digitorum profundus on a straight line between the medial side of the brachial artery at the elbow and a palmaris longus at the wrist. As it courses down the forearm it supplies the pronator teres, flexor carpi radialis, palmaris longus and flexor digitorum sublimis. The interosseous branch innervates the flexor pollicis longus and the flexor digitorum profundus to the index, long and half of the ring fingers. After the palmaris longus inserts into the palmar fascia, the nerve becomes the most superficial structure in the carpal tunnel. In the hand it activates the thenar muscles (recurrent branch) and the two radial lumbricals.* Sensation is given to the volar aspect of the thumb, index and long fingers and usually to the volar aspect of the radical aspect of the ring finger.

The ulnar nerve runs in the ulnar notch then makes the two heads of the flexor carpi ulnaris. It courses down the forearm in the shadow of the flexor carpi ulnaris innervating part of the flexor digitorum profundus. Passing radial to the pisiform bone it divides into a sensory and a motor branch. The motor branch ducks to the skeletal plane to innervate all of the small muscles of the hand except the thenar musculature and the two most radial lumbricals. At times, in its competition with the median nerve, the ulnar nerve can also innervate these latter muscles. The sensory element, if usual, innervates the small and the ulnar half of the ring fingers (Figs. 1-5).

The radial nerve, above the elbow, lies between the brachialis and the brachio-radialis. At the elbow it divides into its deep motor branch which perforates two layers of the supinator innervating this muscle and, after it passes through the muscle, the finger extensors. The sensory branch continues down the forearm as a satellite to the brachioradialis which it perforates. It becomes superficial proximal to the wrist. At the wrist the sensory branch is important since injury to the nerve here frequently causes a troublesome neuroma (Fig. 6, 7).

Pathophysiology

Nerve conduction can be interrupted in various ways. External pressure, as in a tourniquet palsy, carpal tunnel syndrome, pisiform ganglion syndrome, etc. can rob a nerve if its ability to conduct impulses. Relieving the nerve from the pressure usually results in recovery. This recovery is generally shorter in time and more complete than recovery from a laceration of a nerve with subsequent nerve suture.

Strangulation is a phenomenon similar to pressure. Freeing a nerve from a scar bed frequently results in immediate return of sensation and fairly rapid return of motor function although the ultimate return of motor function and the degree of sensor recovery is often incomplete. This "immediate" return of sensation may be explained on the basis of interruption of axoplasmic flow

*The median and ulnar nerves compete for their fields in the hand; e.g. Figure 1 shows the motor fibers to the three radial lumbricals being supplied by the median rather than the median supplying only the most radial two lumbricals.

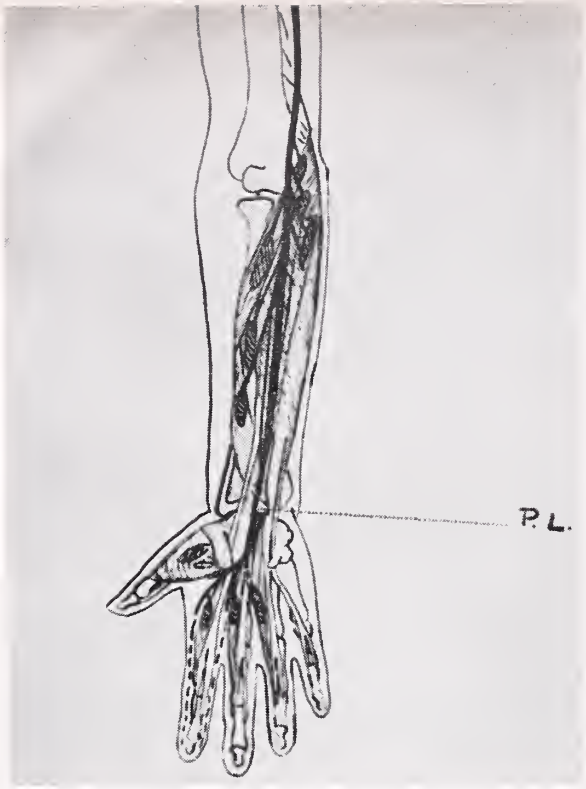


Fig. 1 — Note relationship of the median nerve to the brachial artery and palmaris longus as well as its course sandwiched between flexor digitorum profundus and flexor digitorum sublimis.

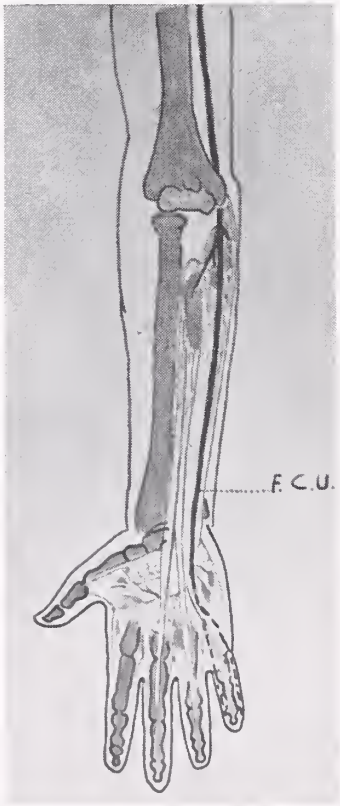


Fig. 2 — The course and distribution of the ulnar nerve on the volar aspect of the forearm. Note the sensory innervation of only the small finger in this particular drawing.

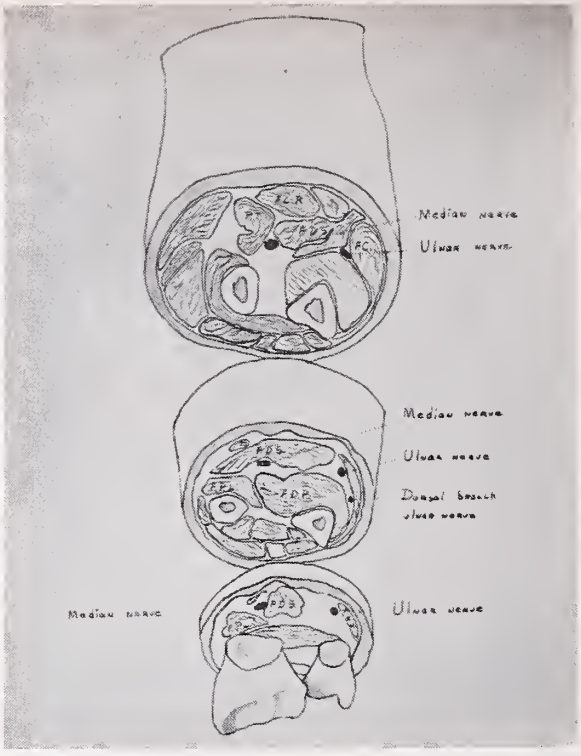


Fig. 3 — The path of the median and ulnar nerves in cross sections of the forearm. Note the median nerve between the flexor digitorum sublimis and profundus and the ulnar nerve running in the shadow of the flexor carpi ulnaris. The dorsal sensory branch of the ulnar nerve arises well above the wrist, perforates the flexor carpi ulnaris and brings sensation to the ulnar portion of the dorsum of the hand. It lies adherent to the forearm fascia.

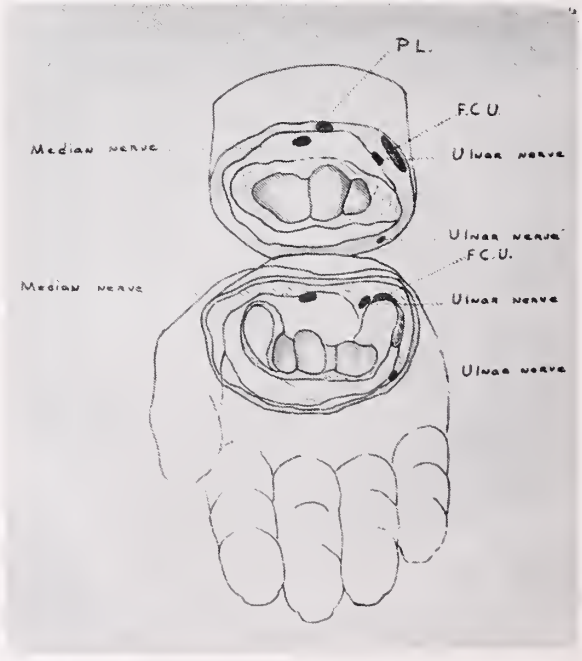


Fig. 4 — Cross section through the carpus shows the satellite relationship of the median nerve to the palmaris longus and of the ulnar nerve to the flexor carpi ulnaris.

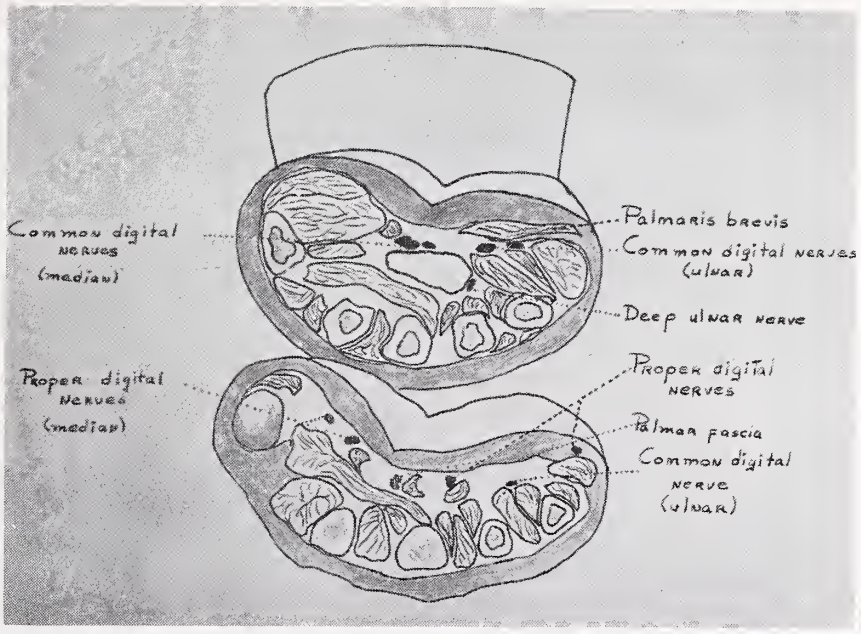


Fig. 5 — Sections through the proximal and middle palm demonstrate the palmar fascia overlying the digital nerves except in the hypothenar eminence and the proximal location and depth of the motor branch of the ulnar nerve.

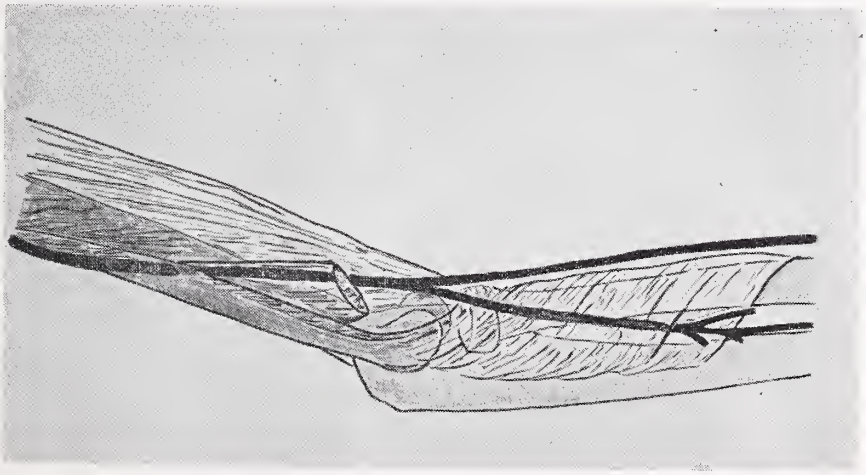


Fig. 6 — The division of the radial nerve into its motor and sensory branches at the elbow. The motor branch is sandwiched between two layers of the supinator.

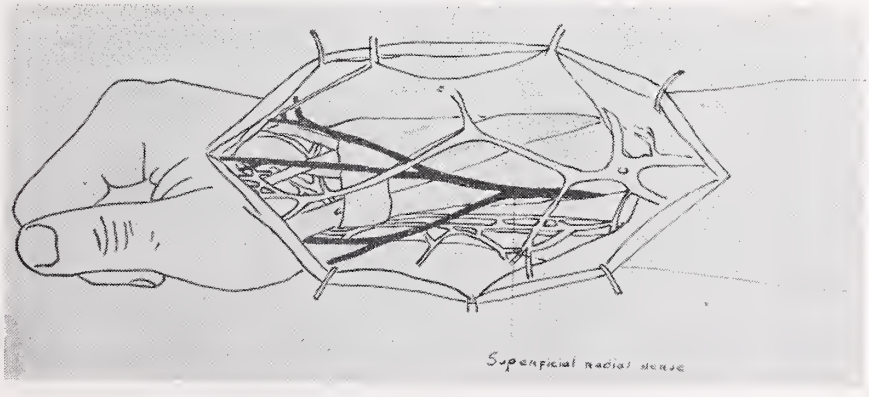


Fig. 7 — The sensory radial nerve at the wrist.



Fig. 8 — Nerve suture is done. Guide sutures at 180 degrees to each other aid in orientation and permit rotating the nerve so that all parts of the sheath can be sutured.

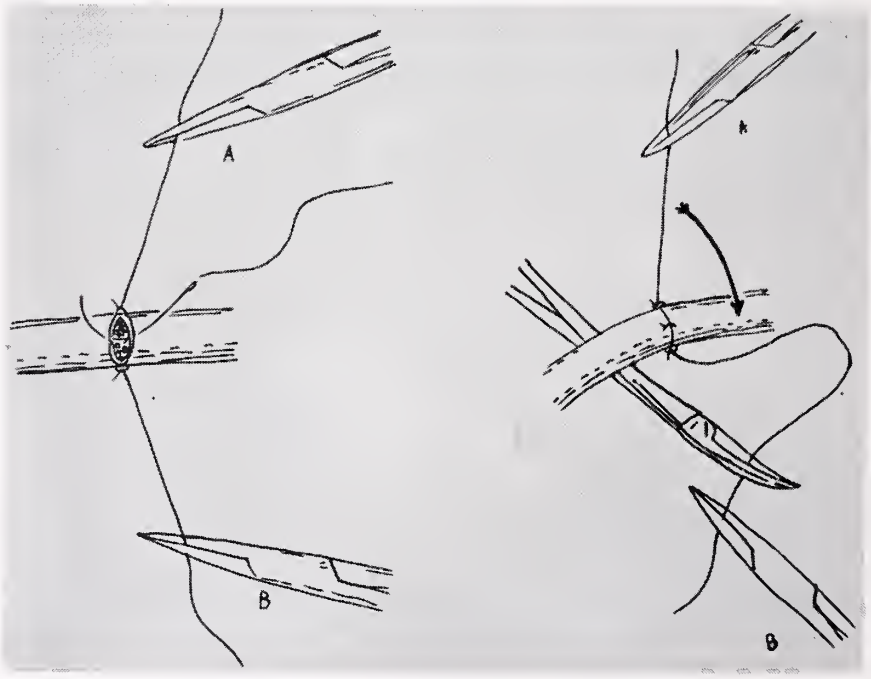


Fig. 9 — The nerve is rotated by the guide suture.

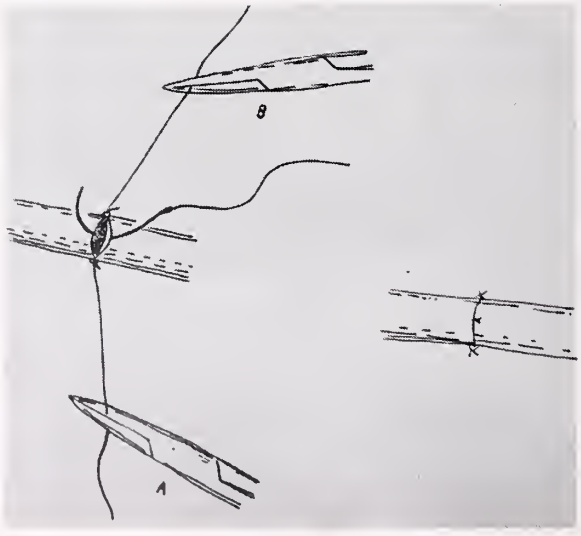


Fig. 10 — The completed suture.

by scar or of low oxygen concentration not permitting repolarization of the nerve membrane.

Interruption of continuity of a nerve may be "clean" as by glass, razor blade, etc. or blunt as by a spinning bullet. Blunt lacerations crush and fray the fibers of a nerve similar to a rope which is pulled beyond its breaking point.

Pathophysiologic principles should guide nerve repair.

Pressure or strangulation of the nerve should be relieved. In repairing clean lacerations axon to axon repair is the ideal. This should be done with either magnifying loupes, glasses or a dissecting microscope. It is vital to connect appropriate "long distance lines." We must not bury material between the axons. A "suturoma" interferes with conduction. Dead space between the axons will permit the formation of a hematoma to form an impenetrable wall of scar. The sheath should be closed completely to prevent the migration of axons out of the limiting membrane (Figs. 8-10).

Nerves should be laid back into a good bed since scar tissue will strangle a nerve. One should try to strip a nerve as little as possible in order to maintain as much blood supply as is feasible. Gentle traction on a nerve is not harmful but forceful pulling may fray axons.

Irradiated nerve grafts surrounded by Millipore have been used in nerve repair. Millipore is a synthetic "cloth" whose pores are small enough to prohibit the entrance of fibroblasts but yet large enough to permit the entrance of tissue fluids. It is theorized that this permits nutrition and excludes fibrous tissue scarring. We all await the follow-up studies on the results of the protection of nerve grafts and nerve suture lines from fibroblastic invasion by the use of Millipore.

The argument as to whether one should carry out primary or secondary suture of nerve continues. Principles should dictate the procedure. In a laceration which is as clean as can be made by the scalpel there is no reason why the nerve should not be repaired primarily. In contrast where a nerve has been crushed, stretched or both and the ends of the nerve are "non-tidy" and hemorrhagic it is difficult to know how much of the nerve is viable. If non-viable nerve enters a suture line an intraneural scar that prohibits nerve conduction results. Therefore, if "non-tidy" nerve is found, secondary rather than primary suture is indicated.

In those cases where delayed repair is carried out or the primary repair is unsuccessful one may find a neuroma incontinuity (intraneural neuroma). Extraneural scarring is frequently found. One way of doing an intra and extraneural neurolysis is to inject saline with a tuberculin needle. This spreads one axon from the other. The technique is also useful in differentiating axons from scar. Axons will "conduct" the water: the nerve balloons out in a characteristic fashion; scar tissue will not permit the flow of saline.

A sometimes problem in peripheral nerve repair is the overcoming of defects. By freeing the median nerve into the upper arm dissecting off the motor branches with the back of the scalpel as one would the stem of a leaf from a branch, by transferring the nerve anterior to the pronator teres, or by flexing the elbow and flexing the wrist one can overcome a distance of four inches in the forearm.

Length can be obtained in the ulnar nerve in the forearm by transposing the nerve anterior to the elbow. One can free up the branches to the flexor carpi ulnaris and can then transfer the nerve on this hinge anteriorly. By flexing the elbow and the wrist, about four inches of lost length of the ulnar nerve can be recovered.

Loss of radial nerve is less of a problem since tendon transfers function well to substitute for absence of motor radial nerve function.

Prior to attempting suture one should estimate the amount of neuroma that has to be excised and the amount of lengthening that you can get. If the distance is more than can be overcome at the time, the nerve should be freed up with the neuromata in place. The neuromata can then be sutured to each other with the elbow and wrist in flexion. Gradual, very judicious, extension of elbow and wrist will permit more lengthening without fraying of the nerve. At a second operation the neuromata can be excised and good nerve tissue sutured to good nerve tissue.

Since the funicular pattern of nerves changes,

a greatest constant length in the median nerve being five millimeters, with resection of more nerve the chance of a good result is less likely.[1]

Upon occasion where there has been bony damage with nonunion of radius, etc., shortening of the bones of the forearm will give further relative nerve length.

When there are defects in median and ulnar nerves in the forearm in the order of six-eight inches a type of repair can still be done. The distal ends of the proximal portions of the nerves are sutured to each other and the ulnar nerve is then cut above the elbow. At the end of four-six weeks the ulnar nerve has attained an autogenous blood supply from the median nerve to which it has been sutured. It can then be freed up and swung down into the forearm to be sutured to the distal median nerve. Some tactile function can thereby be restored.[2]

Prognosis

Return of digital nerve function after its interruption in children under 10 is spontaneous. Perhaps the age of spontaneous recovery of digital nerve function is greater than 10 but I have no guide upon which to make this statement. I have had the experience of exploring hands in children where digital nerves had been interrupted or completely removed and where the sensation including tactilegnosis* in the hand was entirely normal. In the adult after suture even if sensation to light touch returns tactilegnosis does not[3].

The level of laceration is important in the return of function. Axon to axon repair is vital so that proper impulses may go down the proper channels. The more proximal the interruption of a nerve the more mixed the nerve and the greater the chances of improper canalization. The longer the distance the axon has to travel the greater the period of time which must elapse. The more time that elapses the more fibrosis occurs in the muscles, the goal of the motor axons. The more nerve that has to be resected the poorer the prognosis because of the constant changing funicular pattern.

*Tactilegnosis indicates sensation that permits function; e.g. picking up coins, buttoning a coat, use of the hand unguided by vision. This modality parallels two point discrimination (Weber test).

The median nerve is mainly sensory in the forearm and one expects good generation of sensory function except tactilegnosis. Return of function to the thenar muscles is frequently poor necessitating substitution by tendon transfer.

Repair of the ulnar nerve after its division at the pisiform bone in the motor and sensory branches is good since we are dealing with two "pure" nerves. However, proximal to this area the regeneration is not so good although one can usually restore protective sensation along the ulnar aspect of the hand. Return of intrinsic function is exceptional.

The radial nerve beyond the elbow is pure motor or pure sensory. Results of suture are good. However, one usually uses tendon transfers to restore the function of the radial nerve.

The usual rate of regeneration of a peripheral nerve is considered to be from one-two millimeters a day. However, sensory improvement in adults continues up to five years; in children up to two years[5].

Summary

This paper outlines and illustrates the anatomy of the peripheral nerves of the forearm. The pathophysiology of the interruption of nerve conduction is detailed and related to treatment of peripheral nerve injuries. Technical points such as the lysis of intra and extraneural neuromata, overcoming of nerve defects, pedicle nerve grafts and bulb suture are listed. The technique of nerve suture is illustrated. The prognosis in nerve repair depends on the age of the patient, the level of interruption, the type of interruption, the nerve involved and the amount of lost nerve structure which has to be resected.

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Severe Thyrotoxicosis (Thyroid Crisis)

Jerry M. Earll, Maj.,* MC, and Herbert B. McDonald, Capt.,* MC, *El Paso*

The development of effective medical therapy for treatment of thyrotoxicosis has greatly reduced the incidence of thyroid crisis. Thyroid crisis was the most frequent cause of death associated with thyroid surgery prior to the availability of present therapy. Now it is most commonly seen in the medical ward and represents a true medical emergency associated with a 25 per cent mortality rate.[1] In recent years there has been a paucity of clinical articles on thyroid crisis, probably because there have been no new major developments in therapy since the introduction of steroids and reserpine. Basic research has continued to offer new concepts about the biochemistry of the disease and its therapy. The importance of prompt and appropriate therapy of this disease and the incorporation of more recent research developments into our understanding of the disease and its management justify an additional case report and discussion at this time.

Case Report

A 41-year-old Caucasian man was admitted to the hospital on January 18, 1965, with chest pain. He displayed considerable mental confusion, making his history unreliable. Supplemental history from the relatives confirmed abnormal behaviour for at least the past 10 months. Several syncopal episodes and a 40 pound weight loss had been noted. He had complained of nervousness, frequent episodes of trembling, and attacks of painless tachycardia associated with dyspnea. There was no family history of thyroid disease. Physical examination revealed a moderately disoriented, distressed individual with pulse 140, B.P. 160/100, temperature 100°F (37.8° C), respirations 30 per minute. The thyroid gland was moderately enlarged and there was a bruit over the gland. The neck veins were slightly distended when the patient was at a 45° angle. The precordium was hyperdynamic with the point of maximal impulse two centimeters lateral to the mid-clavicular line. The liver was enlarged and palpable six centimeters below the right costal margin. There was no peripheral edema. The pulses were bounding, the deep tendon reflexes were hyperactive, and coarse and fine tremors were demonstrated in all extremities. Venous pressure was 130 mm. of saline and a decholin arm-to-tongue circulation time was 10 seconds. Hemogram, urinalysis, serum electrolytes, liver and renal function studies were normal except for a mild relative lymphocytosis. Serum cholesterol was 130 mg/100 ml and the protein-bound iodine 13.6 ug/100 ml. A four-hour radioactive iodine uptake was 80 per cent. Twenty-four hour urinary catecholamines revealed a normal value of 40 mcg. Electrocardiogram was abnormal, revealing a sinus tachycardia with rate of 135 per minute and nonspecific ST-T changes. Chest x-ray was normal.

No evidence of infection could be elicited, and the patient's impending thyroid storm was treated promptly with reserpine I.M., 0.5 mg test dose followed by 2.5 mg q six hours, Thorazine 75-100 mg q six hours, sodium luminal 100 mg b.i.d. orally, intravenous five per cent glucose and water, hydrocortisone sodium succinate 100 mg immediately and 100 mg daily by intravenous drip, oxygen therapy in a cool tent, saturated solution of potassium iodide five drops t.i.d. and propylthiouracil 500 mg q six hours. The patient gradually improved and he reached a euthyroid state in April, 1965. There had been no evidence of myocardial infarction. Following a three day withdrawal of propylthiouracil, the 24 hour radioactive iodine uptake returned to 99 per cent. He

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was treated with 11.0 millicuries of radioactive iodine and remained euthyroid for the next three months.

Discussion

The case presented is a typical clinical picture of severe thyrotoxicosis, and emphasizes the commonly associated mental confusion which often results in serious delay in the patient's presentation for therapy. Terms such as thyroid crisis and thyroid storm are recognized by all clinicians as defining life-endangering emergencies. They carry with them the unfortunate necessity of requiring a definition, and while few would dispute their usefulness in selecting an important and seriously ill group of thyrotoxic patients, adherence to rigid criteria sometimes results in failure to recognize the seriously ill patient and subsequent delay in adequate treatment. The state of the thyrotoxic patient is a clinical assessment not necessarily related to any laboratory parameters, and this is why we prefer the term "severe thyrotoxicosis." Severe thyrotoxicosis differs from thyrotoxicosis by degree, the signs being panoramic and intensified to life-threatening proportions. Waldstein et al's[1] criteria are excellent, and thyroid storm was considered present whenever accentuated signs and symptoms of hyperthyroidism were accompanied by a fever of 100°F or greater and marked tachycardia. The point is well made that fever does not occur in uncomplicated thyrotoxicosis. It is interesting to note that the body temperature control and thyroid control areas are in close proximity in the hypothalamus. Few body systems escape effects of the exacerbated hypermetabolic state; and central nervous system, gastrointestinal, hepatic, and cardiovascular manifestations are prominent.

The onset varies from extremely sudden to very gradual, and precipitating factors are multiple, with infection, surgical procedures, and trauma being the most common. It is known that thyroxine produces uncoupling of oxidative phosphorylation associated with structural changes in mitochondria — suggesting thyrotoxicosis at the mitochondral level is a disease of defective energy transfer.[2] The transfer of energy obtained from oxidation to phosphorylative bond energy has been termed "coupling," and interference with this transfer of energy can result in a high oxidative rate with little useful energy output. The increased tissue metabolism could result in relative tissue anoxia and the accumulation of precursors of adenosine triphosphate (ATP). Adenosine mono, di, and triphosphate are potent vasodilators which act primarily on small vessels, particularly the arterioles. This vasodilation may contribute to the high cardiac output and wide pulse pressure seen in hyperthyroidism, for if there is ample widespread vasodilatation, results on the circulation will be similar to a large arteriovenous shunt or aortic insufficiency. With decreased resistance in some of the vascular bed, compensatory mechanisms occur. Reduced renal blood flow, the retention of sodium, and expansion of the blood volume help to prevent shock. The heart-muscle mitochondria are particularly susceptible to uncoupling,[4] and the requirement of a high cardiac output with inefficient energy production makes cardiac decompensation a major threat to progressive thyrotoxicosis. Congestive heart failure associated with thyrotoxicosis is frequently resistant to digitalis. The role of digitalis therapy in thyrotoxicosis is a questionable one. In this extremely dynamic state, cardiac decompensation can occur very rapidly.

The exaggerated sympathetic responses of hyperthyroid patients is reduced with reserpine[5] or guanethidine.[6] Guanethidine selectively interrupts adrenergic reflexes at their neuroeffector junction and both drugs deplete catecholamines in some tissues. In view of the earlier discussion of interference with the production of ATP, it is interesting to note that ATP and adenosine diphosphate (ADP) enhance the uptake of catecholamines from incubation fluid and retard the spontaneous loss of noradrenaline from a suspension of adrenergic nerve granules.[7] It would seem that if this is true in vivo, it would make the hyperthyroid individual more sensitive to drugs depleting the catecholamines. It has been pointed out, however, that hyperthyroid patients are actually more resistant to these drugs.[8] The mechanism of action of these agents is not well worked out at an intracellular level, and though there is some controversy, there is good evidence that catecholamine excretion is not significantly altered in hyperthyroidism.[9]

The question of relative adrenal insufficiency is frequently raised in the severe thyrotoxic patient. The simultaneous occurrence of Addison's disease and thyrotoxicosis has been reported often enough to suggest more than a chance association, and

both antithyroid and antiadrenal antibodies were recently identified in a patient's serum.[10] Cortisone probably has some effect at a cellular level and it inhibits the release of thyroxin. Its use is still quite empirical, as true adrenal insufficiency is probably much rarer than originally believed. In spite of this, in this life-threatening condition, steroids should be used.

As in most medical emergencies, the prevention of severe thyrotoxicosis by earlier diagnosis and treatment is of major importance. Although some clinicians would criticize reserpine because of the possibility of further mental deterioration, the fact remains that it is effective in counteracting adrenergic manifestations and also a good sedative. Following an initial test dose of .25-.5 mg parenterally, 2.5 mg every 4-6 hours is usually adequate. Guanethidine is used in doses of 50-150 mg/day, using caution to avoid serious side effects. It does not possess the required sedative effect of reserpine and additional sedation is usually required. If barbiturates are selected, one must be alert for the occasional patient who reacts adversely; if morphine is selected, there is danger of respiratory depression. Hydrocortisone in doses of 100-300 mg daily is given intravenously or intramuscularly. Equivalent or higher doses of prednisolone may be helpful without the marked fluid retaining properties of hydrocortisone. If the patient is already edematous, salt restriction is indicated. Supportive therapy includes maintenance of hydration with glucose and saline, oxygen therapy for relief of anoxia, and control of hyperpyrexia with any effective method from sponging to a hypothermia blanket. These are the therapies that make the immediate difference in prognosis. Iodine and propylthiouracil do little to alter the immediate situation, but are extremely important in the eventual control of the disease. Therapy is introduced promptly and iodine can be given intravenously or orally depending upon the situation. In a conscious patient, we prefer oral saturated solution of potassium iodide in doses of five to 15 drops daily. Propylthiouracil is given in doses of 250-500 mg every six hours, and if the patient is unconscious, this is given by intubation. Any precipitating event which is treatable such as an infection must be dealt with promptly. Definitive therapy with either radioactive iodine or surgery is indicated much later, when the patient is under good control.

Summary

A typical patient with thyroid crisis is presented. Too rigid criteria for the diagnosis of thyroid crisis can result in delay in treating a life-threatening condition, and the term severe thyrotoxicosis may be useful in this respect. Although there has been no major change in the therapy of severe thyrotoxicosis since the introduction of steroids and reserpine, clinical investigation and research have continued to contribute to our understanding of the disease and its management. A discussion of some of these aspects is included and an attempt has been made to relate them to the disease as the clinician sees it.

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Dr. Frank A. Rowe Elected President Southwestern Medical Ass'n

Dr. Frank A. Rowe, Albuquerque, was elected President of the Southwestern Medical Association at its 47th Annual Meeting in El Paso, November 4-6, 1965.

Other new officers are Dr. Laurance N. Nickey, El Paso, President-Elect, Dr. Zigmund W. Kosicki, Santa Fe, Vice-President, and Dr. Sol Heinemann, El Paso, Secretary-Treasurer. Dr. Clement C. Boehler, El Paso, was the Retiring President.

Members of the Executive Committee, elected at the meeting, are Dr. Boehler, Dr. Robert F. Boverie, El Paso, Dr. Louis W. Breck, El Paso, Dr. John S. Carlson, Phoenix, Dr. C. W. Carroll, Las Cruces, New Mexico, Dr. Homero Galindo, Juarez, Chihuahua, Mexico, Dr. Carlos Tapia, Hermosillo, Sonora, Mexico, and Dr. J. Warner Webb, Jr., Tucson.

Speakers at the meeting were Dr. Irvin E. Hendryson, member of the AMA Board of Trustees, who talked on "Medicare: Its Effect On Your Practice"; and Dr. Arthur A. Clinco, Dr. Donald G. Mulder, Dr. John F. Murray, and Dr. Justin J. Stein, all from the University of California at Los Angeles School of Medicine, which presented the scientific program.

Dr. Rowe was born in Albuquerque and received a B.S. from the University of New Mexico and his M.D. from the St. Louis University School of Medicine. He interned at St. John's Hospital in St. Louis, where he also took a Surgical Residency.

He then served in the U.S. Army Medical Corps, with an overseas tour of duty in Kassel, Germany, where he was Ward Surgeon and Chief of the Surgical Service in the 386th Station Hospital.

He began the private practice of Medicine in Albuquerque in 1947 and was associated with Dr. William H. Woolston, with whom he served a Surgical Preceptorship until Dr. Woolston's death in 1957.

He was President of the Bernalillo County Medical Association in 1959 and a member of its Board of Governors from 1958-60. He is a member of the Southwestern Surgical Society and the American College of Abdominal Surgery. He is a former Chief-of-Staff of St. Joseph's Hospital.

He is a Knight of St. Gregory. He and his wife are the parents of four daughters and two sons.



NEW SWMA OFFICERS — Dr. Frank A. Rowe of Albuquerque (second from left) was elected President of the Southwestern Medical Association at the group's 47th Annual Meeting in El Paso, Nov. 4-6, 1965. Left to right are Dr. Gordon L. Black, El Paso, General Chairman; Dr. Rowe; Dr. Sol Heinemann, El Paso, re-elected Secretary-Treasurer, and Dr. A. Robert Nering, El Paso, Scientific Program Chairman. Not shown are Dr. Laurance N. Nickey, El Paso, President-Elect, and Dr. Zigmund W. Kosicki, Santa Fe, who was re-elected Vice-President. Retiring President was Dr. Clement C. Boehler of El Paso.



MEDICARE SPEAKER — Dr. Irvin E. Hendryson, second from right, Denver, member of the American Medical Association Board of Trustees, spoke on "Medicare: Its Effect on Your Practice" at the Annual Meeting of the Southwestern Medical Association in El Paso. With him are, left to right, Dr. John F. Murray and Dr. Donald G. Mulder, guest speakers from the University of California at Los Angeles Medical School of Medicine, and Dr. Clement C. Boehler, El Paso, Immediate Past-President of the Association.

N.M. Medical Society Holds Interim Meet

The New Mexico Medical Society at its Interim Meeting in Clovis, New Mexico, November 5-6, 1965, asked that medical care service for all persons on New Mexico Department of Public Welfare rules be transferred to the New Mexico Department of Public Health because of the DPH professional medical competence.

The Society's House of Delegates requested the Governor of New Mexico to direct the DPW to contract with the DPH to administer all medical services and to transfer such responsibility and appropriations to the DPH. The Society took the position that the DPW should be concerned solely with non-medical assistance programs.

Dr. Robert P. Beaudette, Raton, President, said the Society "is disturbed that Welfare patients are being treated as 2nd class citizens instead of as sick people".

The Society approved a continuing Medical Education Program for New Mexico physicians through color, closed-circuit TV with two-way communication.

Gary Anderson, Ph.D., Dean of Eastern New Mexico University, was the banquet speaker. Speakers on the scientific program were Dr. Solomon Papper, Professor of Medicine, University of New Mexico Medical School; Dr. William Lester, Chief of Chest Medicine, National Jewish Hospital, Denver; Dr. Joseph Messer, Professor of Medicine, Tufts Medical School, Boston; and Dr. Robert Whang, Assistant Professor of Medicine at the University of New Mexico Medical School.

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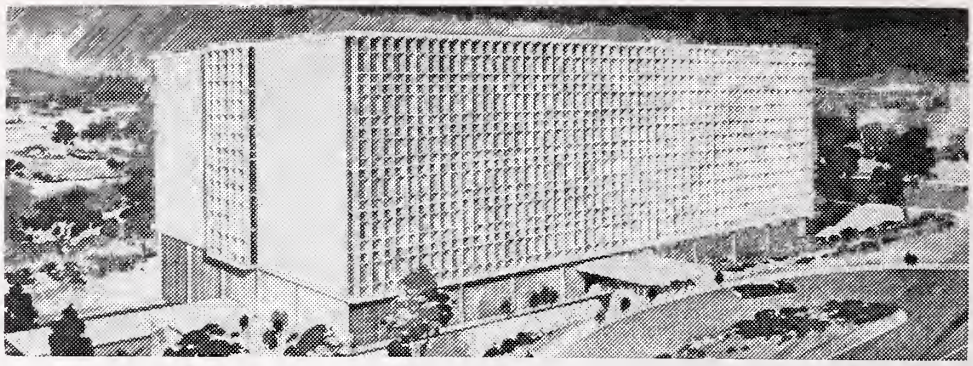
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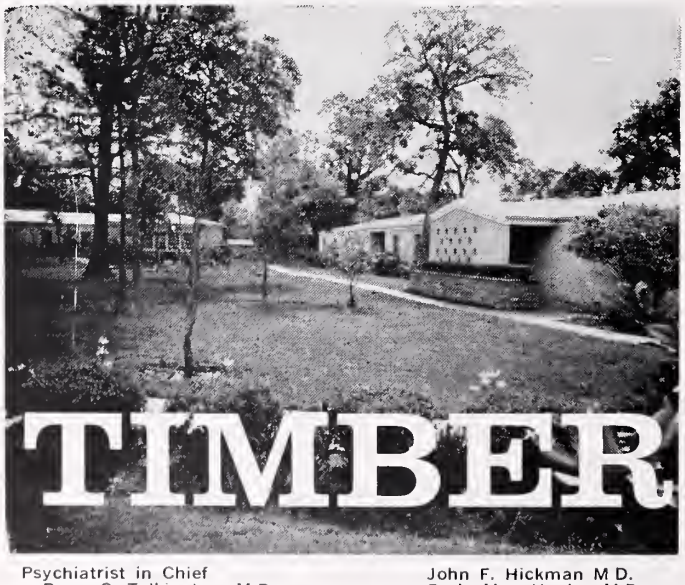
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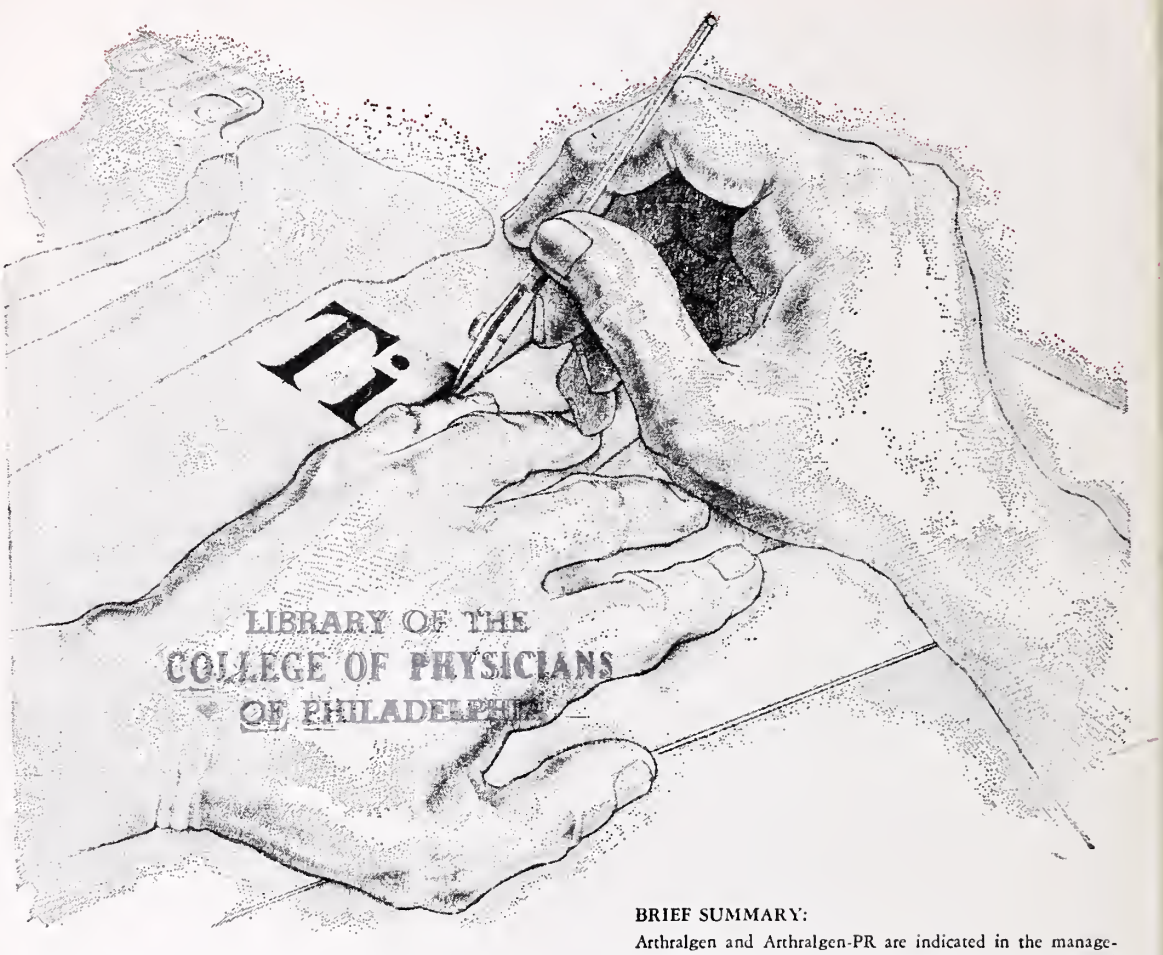
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